University of Alberta

# The Effect of High-Intensity Resistance Training on the Hormonal and Strength Responses in Healthy Elderly Males

by

Michael Jon Edward Souster

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Masters of Science

Department of Physical Education and Recreation

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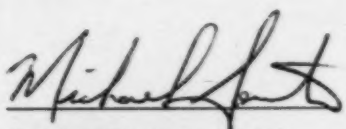

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# ABSTRACT

The purpose of this study was to determine whether resistance training would elicit a change in the anabolic hormonal status of elderly males. Twenty healthy males between the ages of 62-76 were matched for strength and randomly assigned into a control group and a resistance training group. The resistance training group was required to complete a 16-week, high-intensity, periodized program. Total testosterone (TT), free testosterone (FT), growth hormone (GH), urinary cortisol, estradiol and sex hormone-binding globulin (SHBG) concentrations were determined. There was an increase in upper and lower body strength after 16 weeks of resistance training ($p<0.05$). There was a decrease in TT after the 16-week training period ($p<0.05$). FT, GH, urinary cortisol, estradiol and SHBG were unchanged with resistance training. These results suggest that heavy resistance training was effective at increasing upper and lower body strength in absence of an enhanced hormonal response in elderly males.

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## LIST OF SYMBOLS, NOMENCLATURE OR ABBREVIATIONS

| | |
|---|---|
| TT | Total Testosterone |
| FT | Free Testosterone |
| GH | Growth Hormone |
| SHBG | Sex Hormone-Binding Globulin |
| IGF-I | Insulin-Like Growth Factor I |
| MHC | Myosin Heavy Chain |
| PFK | Phosphofructokinase |
| LDH | Lactate Dehydrogenase |
| Hb | Hemoglobin |
| Hct | Hematocrit |
| 1RM | One Repetition Maximum |
| mRM | Multiple Repetition Maximum |
| CT | Control Group |
| RT | Resistance Trained Group |
| RIA | Radioimmuno Assay |
| ANOVA | Analysis of Variance |
| LBM | Lean Body Mass |
| CSA | Cross-Sectional Area |
| SR | Sarcoplasmic Reticulum |

# CHAPTER 1

# INTRODUCTION

## Background

The elderly are the fastest growing segment of the population in Canada and the United States. In 1991, individuals over the age of 65 years accounted for 12% of the Canadian population. The changing demographics in Canada have alluded that this number will to grow to 20% by the year 2031. These statistics imply that in the next four decades, the Canadian senior citizen population will increase from 3.2 million to more than eight million (Elliot, 1996). In addition, the American College of Sports Medicine (1998) reports that by the year 2030, the number of individuals over the age of 65 years will reach 70 million in the United States alone. This trend in the population has heightened the interest in aging research.

As the number of people living to an older age increases, the ability to remain functionally independent is, unquestionably, a primary objective for these older individuals. In recent years, both research and the media have focused on the importance of regular physical activity to improve health, functional capacity, and for maintaining quality of life in the aging adult. Moreover, health professionals have expressed an increased emphasis on the importance of aerobic activity to improve health and well-being in the elderly. Some of these so-called aerobic activities include walking, jogging, swimming, hiking and cycling. Even though it has been many years since research initially demonstrated that aerobic exercise induces cardiovascular health benefits in the older adult (deVries, 1970), it has been reported that aerobic exercise does not compensate for age-associated muscle wasting (Klitgaard et al., 1990). If participation in aerobic activity can not prevent the reductions in muscle volume and strength associated with aging (Klitgaard et al., 1990) then some activities of daily living may be compromised (even for those individuals physically active in aerobic exercise). Thus allowing the threshold for dependency to be approached or surpassed. Therefore, it is

very important for aged individuals to ascertain an intervention that, at the least, retains the minimal level of muscular fitness that is critical for independence.

Human skeletal muscle begins to atrophy at the age of 25 years in men, but this loss is only 10% for the next 25 years. After the age of 50 years, an additional 30% loss in muscle mass occurs by the age of 80 years (Lexell et al., 1986). In a review conducted by Rodgers and Evans (1993), it is postulated that this decrease in muscle size with aging could account for much of the reduction in muscle strength. Furthermore, in addition to the frequently observed reduction in muscle volume, there is also a decrease in motor unit number, activation, and synchronization that occurs with aging. All of these decreases contribute to the reduction in strength. Nonetheless, age-associated muscle wasting is not recognized as a syndrome by many physicians because the progressive muscle weakening is accepted as normal since its onset is so gradual (Tseng et al., 1995).

Whether age-associated muscle wasting is considered a syndrome or not, the progressive loss of muscle mass and strength with aging is a major public health concern. Tseng et al. (1995) stated that the loss of strength prohibits independent living for the aged and that this contributes to a major financial drain on the health care system. This financial drain can be partly attributed to both acute and chronic problems exacerbated by lack of muscle strength in the elderly. Acute care costs of fall related injuries due to lack of strength are estimated to be $10 billion per year in the United States (Tinetti et al., 1994). Chronic problems such as insufficient strength and endurance to perform activities of daily living add a further $7 billion per year in health care costs (Rowlands and Lyons, 1991). Unless some sort of intervention is implemented, this financial cost resulting from muscular strength loss, is of particular concern as these figures can only be expected to increase due to the growth of the senior population.

In addition to age-related changes in function and morphology of skeletal muscle, the aging process is also associated with hormonal changes. Contrary to the female reproductive system, men do not ordinarily experience the rapid decline or arrest of reproductive capacity affiliated with aging. Nonetheless, the aging process is related with a number of hormonal changes such as a decrease in total (TT) and free testosterone (FT) concentrations (Gray et al., 1991), as well as growth hormone levels (GH) (Ho et al., 1987). At the same time, cortisol (Barton et al., 1993), estradiol (Drafta et al., 1982) and

the principal testosterone carrier protein, sex hormone-binding globulin (SHBG) (Gray et al., 1991) have a tendency to increase with age. These trends in hormonal changes may have a direct effect on the lean body mass of elderly individuals.

The atrophy of lean body mass and diminished muscle function, characteristic of the elderly, results partially from diminished secretion of GH (Rudman, 1985), and these changes also occur in conjunction with a decrease in serum testosterone concentrations (Nankin et al., 1986). Studies investigating testosterone and GH administration in elderly individuals demonstrate an increase in skeletal muscle strength and protein synthesis (Rudman et al., 1990; Urban et al., 1995). However, excess GH can cause impaired glucose tolerance, hypertension, articular and muscular pain, carpal tunnel syndrome and fluid retention (Marcus et al., 1990). In addition, the cost of hormone supplementation may add to the already high health care costs for the elderly. Therefore, short and long term supplementation of these hormones may not be the reasonable solution to the hormonal and muscular changes associated with aging.

Investigators have demonstrated that resistance exercise may be an effective method to compensate for the age-related changes in function and morphology of aging human skeletal muscle (Moritani & deVries, 1980; Frontera et al., 1988; Fiatarone and Evans, 1993; Roman et al., 1993). In addition, several recent investigations involving young, sedentary individuals have shown that high-intensity resistance training may increase total basal testosterone levels (Hakkinen et al., 1985; Busso et al., 1990) and GH release after resistance exercise (Vanhelder et al., 1984; Craig & Kang, 1994). However there is a paucity of information regarding the hormonal responses to high-intensity resistance training in the elderly population, and the impact that the benefits of resistance exercise has on the quality of life and health in the elderly may in fact be greater than any other form of exercise.

## Statement of the Problem

There is a paucity of information regarding the hormonal responses to high-intensity resistance training in the elderly population.

## Primary Question

Does a 16-week high-intensity resistance-training program produce statistically different basal hormonal concentrations in total testosterone, free testosterone, growth hormone, estradiol, cortisol, and the hormone carrier protein, sex hormone-binding globulin?

## Secondary Question

Does a 16-week high-intensity resistance training program produce statistically significant changes in upper and lower body muscular strength?

**Primary Hypothesis**

There are significant differences between pre- and post-training dependent measurements of basal plasma concentrations of total testosterone, free testosterone, growth hormone, estradiol, sex hormone-binding globulin and basal urinary cortisol concentrations.

**Secondary Hypothesis**

There are significant differences between pre- and post-training dependent measurements in upper and lower body muscular strength.

**Significance of the Study**

Age-associated muscle wasting is a syndrome that effects the vast majority of the elderly population. As the performance of daily living tasks becomes diminished, the ability of elderly individuals to remain independent and continue to be functional members of society throughout life is also reduced. Therefore, improving muscle protein anabolism in the elderly is essential to maintain physical function and independence. Both resistance exercise and hormone replacement therapy have been proposed as protein anabolic interventions in the elderly. If resistance exercise can be shown to be an effective means of increasing the anabolic hormonal status in elderly individuals, then hormone therapy may not be required. Thus the identification of resistance exercise as an anabolic intervention may possibly result in a growing number of elderly individuals who can remain independent into later years and, at the same time be less dependent on the health care system.

## References


American College of Sports Medicine. (1998). Position stand on exercise and physical activity for older adults. Medicine and Science in Sports and Exercise, 30 (6), 992-1008.

Barton, R.N., Horan, M.A., Weijers, J.W.M., Sakkee, A.N., Roberts, N.A., & van Bezooijen, C.F.A. (1993). Cortisol production rate and the urinary excretion of 17-hydroxycorticosteroids, free cortisol, and 6β-hydroxycortisol in healthy elderly men and women. Journals of Gerontology, Medical Sciences, 48 (5), M213-M218.

Busso, T., Hakkinen, K., Pakarinen, A., Carasso, C., Lacour, J.R., Komi, P.V., & Kaukanen, H. (1990). A systems model of training responses in elite weight lifters. European Journal of Applied Physiology, 61, 48-54

Craig, B.W. and Kang, H. (1994). Growth hormone release following single versus multiple sets of back squats: Total work versus power. Journal of Strength and Conditioning Research, 8 (4), 270-275.

deVries, H.A. (1970). Physiological effects of an exercise training regimen upon men aged 52-88. Journal of Gerontology, 25 (4), 325-336.

Drafta, D., Schindler, A.E., Stroe, E., & Neacsu, E. (1982). Age-related changes of plasma steroids in normal adult males. Journal of Steroid Biochemistry, 17, 683-687.

Elliot, G., Hunt, M., & Hutchinson, K. (1996). Facts on Aging in Canada. Hamilton: McMaster University, Office of Gerontological Studies.

Fiatarone, M.A. and Evans, W.J. (1993). The etiology and reversibility of muscle dysfunction in the aged. The Journals of Gerontology, 48 (Special Issue), 77-83.

Frontera, W.R., Meredith, C.N., O'Reilly, K.P., Knuttgen, H.G., & Evans, W.J. (1988). Strength conditioning in older men: Skeletal muscle hypertrophy and improved function. Journal of Applied Physiology, 64 (3), 1038-1044.

Gray, A., Feldman, H.A., McKinlay, J.B., & Longcope, C. (1991). Age, disease, and changing sex hormone levels in middle-aged men: Results of the Massachusetts male aging study. Journal of Clinical Endocrinology and Metabolism, 73 (5), 1016-1025.

Hakkinen, K., Pakarinen, A., Alen, M., & Komi, P.V. (1985). Serum hormones during prolonged training of neuromuscular performance. European Journal of Applied Physiology, 53, 287-293.

Ho, K.Y., Evans, W.S. Blizzard, R.M., Veldhuis, J.D., Merriam, G.R., Samojlik, E., Furlanetto, R., Rogol, A.D., Kaiser, D.L., & Thorner, M.O. (1987). Effects of sex and age on the 24-hour profile of growth hormone secretion in man: Importance of endogenous estradiol concentrations. Journal of Clinical Endocrinology and Metabolism, 64 (1), 51-58.

Klitgaard, H., Mantoni, M., Schiaffino, S., Ausoni, S., Gorza, L., Laurent-Winter, C., Schnohr, P., & Saltin, B. (1990). Function, morphology and protein expression of ageing skeletal muscle: a cross-sectional study of men with different training backgrounds. Acta Physiol. Scand, 140, 41-54.

Lexell, J., Downham, D., & Sjostrom, M. (1986). Distribution of different fibre types in human skeletal muscles. Fibre type arrangement in m. vastus lateralis from three groups of healthy men between 15 and 83 years. Journal of Neurological Science, 72, 211-222.

Marcus, R., Butterfield, G., Holloway, L., Gilliland, L., Baylink, D.J., Hintz, R.L., & Sherman, B.M. (1990). Effects of short term administration of recombinant human growth hormone to elderly people. Journal of Clinical Endocrinology and Metabolism, 70 (2), 519-527.

Moritani, T. and deVries, H.A. (1980). Potential for gross muscle hypertrophy in older men. Journal of Gerontology, 35 (5), 672-282.

Nankin, H.R. and Calkins, J.H. (1986). Decreased bioavailable testosterone in aging normal and impotent men. Journal of Clinical Endocrinology and Metabolism, 63 (6), 1418-1420.

Rodgers, M.A. and Evans, W.J. (1993). Changes in skeletal muscle with aging: Effects of exercise training. Exercise and Sports Science Reviews, 21, 65-102.

Roman, W.J., Fleckenstein, J., Stray-Gundersen, J., Alway, S.E., Peshock, R., & Gonyea, W.J. (1993). Adaptations in the elbow flexors of elderly males after heavy-resistance training. Journal of Applied Physiology, 74 (2), 750-754.

Rowlands, D. and Lyons, B. (1991). Financing home care. Baltimore: John Hopkins University Press, 244.

Rudman, D. (1985). Growth hormone, body composition, and aging. Journal of the American Geriatric Society, 33, 800-807.

Rudman, D., Feller, A.G., Nagraj, H.S., Gergans, G.A., Lalitha, P.Y., Goldberg, A.F., Schlenker, R.A., Cohn, L., Rudman, I.W., & Mattson, D.E. (1990). Effects of human growth hormone in men over 60 years old. The New England Journal of Medicine, 323, 1-6.

Tinetti, M.E., Baker, D.I., McAvay, G., Claus, E.B., Garret, P., Gottschalk, M., Koch, M.L., Trainor, K., & Horwitz, R.I. (1994). A multifactorial intervention to reduce the risk of falling among elderly people living in the community. New England Journal of Medicine, 331, 821-827.

Tseng, B.S., Marsh, D.R., Hamilton, M.T., & Booth, F.W. (1995). Strength and aerobic training attenuate muscle wasting and improve resistance to the development of disability with aging. The Journals of Gerontology, Series A, 50A, 113-119.

Urban, R.J., Bodenburg, Y.H., Gilkison, C., Foxworth, J., Coggan, A.R., Wolfe, R.R., & Ferrando, A. (1995). Testosterone administration to elderly men increases skeletal muscle strength and protein synthesis. American Journal of Physiology, 269, E820-E826.

Vanhelder, W.P., Radomski, M.W. & Goode, R.C. (1984). Growth hormone responses during intermittent weight lifting exercise in men. European Journal of Applied Physiology, 53, 31-34.

# CHAPTER 2

# REVIEW OF LITERATURE

## Muscle Physiology: Strength Loss and Skeletal Muscle Alterations with Aging

The decline in muscular strength is a relatively predictable characteristic of the aging process. However, when studying the aging process of skeletal muscle, it is important to remember that the rates of aging are highly variable among muscle groups and individuals. In addition, the relative contributions of biological aging, cumulative diseases, inactivity and nutritional deficiencies, to the decline in muscular strength, remain unclear. Therefore, the study of muscle and the aging process must be handled with discretion, by distinguishing the possible confounding variables involved.

In a study conducted by Larsson et al. (1979), skeletal muscle strength tended to increase up to the age of 30 years, plateau until approximately 50 years of age and thereafter decline slowly. However, as mentioned previously, the amount of strength loss due solely to biological aging is up to question. Although the notion that the loss of strength in the elderly being primarily the result of physical inactivity is still present, Aniansson and coworkers (1983) have suggested that the loss of strength due to biological aging is approximately 3% per annum from age 69 to 80 years. The longitudinal investigation conducted by Greig et al. (1993) however, revealed that there was no significant change in the isometric quadriceps strength of 14 men and women as they aged from 74-81 years. It is important to mention that the subjects in this study reported above average habitual physical activity for their age. Whether strength loss is the consequence of physical inactivity or the outcome of biological aging, it is important to recognize that older skeletal muscle is more inclined to lose strength. A further understanding of age-related muscle changes could enable the development of interventions that may slow the decline of muscle function with biological aging. According to Porter and Vandervoort (1995) and Booth et al. (1994), the principle age-related changes of the neuromuscular system believed to contribute to the strength loss include:

- Morphologic Changes
- Neurologic Changes
- Intrinsic Muscle Property Changes
- Muscle Enzyme Changes

## Morphologic Changes

Age-related loss in skeletal muscle mass has been referred to as sarcopenia. Muscle atrophy is caused by a decrease in muscle fiber number and a decreased fiber cross-sectional area (CSA) (Lexell et al., 1983). Lexell and coworkers (1988) examined muscle volume of the entire vastus lateralis muscle taken at autopsy in 43 previously healthy male individuals age 15-83 years. It was found that between the ages of 24-80 years, an individual loses approximately 40% of total skeletal muscle mass. From 24-50 years of age, 10% of total muscle CSA is lost. Thereafter, muscle atrophy is accelerated so that between 50 and 80 years of age, an additional 30% of total muscle CSA is lost. In addition to skeletal muscle atrophy, the muscle also becomes infiltrated with greater amounts of fat and connective tissue (Lexell et al., 1988). It is believed that these tissues replace the denervated muscle fibers from lost motor units.

An early study conducted by Larsson (1983) reported an increase in the percentage of type I fibers. However, later studies (Sato et al., 1984; Lexell et al., 1988) found an equivalent loss of both type I and type II muscle fibers. Although there is an equivalent loss in the number of type I and type II muscle fibers, it has been observed that there is a prominent decline in CSA of type II fibers, whereas type I fiber CSA is often less effected (Tomonaga, 1977).

In a study conducted by Klitgaard and colleagues (1990a), analysis of myosin heavy chain (MHC) composition in human m. lateralis muscle demonstrated a higher proportion of fibers possessing a coexistence of MHC types I and IIa as well as MHC types IIa and IIb. This indicates an increased coexistence of MHC isoforms in histochemically determined fiber types with aging.

## Neurologic Changes

Neurologic changes, associated with aging, begins at 30 years of age, but significant levels are not detectable until approximately 50 years of age (Tomlinson and Irving, 1977). At this time, demyelinization of spinal and peripheral nerves, and ventral horn cell body loss both contribute to a decline in the number of motor units. Lexell and Downham (1991) present evidence that, with the loss of motor units either a) the muscle fibers from lost motor units are not being reinnervated, or b) some muscle fibers from the lost motor units are reinnervated by another nerve terminal of an adjacent motor neuron. This latter phenomenon is termed collateral nervous sprouting and is supported by both the enlargement of motor unit size and muscle fiber type being transformed from a random spatial arrangement of fibers to fiber-type grouping during the aging process (Booth et al., 1994). Since motor unit recruitment progresses from the small (slow twitch) to large (fast twitch) motor units during exercise, the increasing size of motor units with aging (due to denervation-reinnervation) could account for the increased latent periods and delayed readiness in muscle contraction as larger motor units are substituted for smaller motor units at low force (Nelson et al., 1984). In addition to motor unit loss, profound abnormalities at the neuromuscular junction occur. Other observations that have been reported include a) a gradual withdrawal of the terminal axon and b) a reduction in nerve terminal area (Gutmann and Hanzlikova, 1972; Tomonaga, 1977).

## Intrinsic Muscle Property Changes

The deterioration of the excitation-contraction coupling process may also contribute to decline in force production during aging. It has been observed that rates of force development have been shown to decrease in the elderly (Clarkson et al., 1981). Larsson and Salviati (1989) measured various $Ca^{2+}$ activities in the rat and found that the prolongation of twitch duration in old age is related to both a decrease of sarcoplasmic reticulum (SR) volume and a decrease of the $Ca^{2+}$ pump activity in type II fibers. However, unlike the type II fibers, the SR and the intrinsic properties of SR were not affected by age in the type I muscle fibers, thus suggesting that other factors are

responsible for the prolongation of isometric twitch duration in type I fibers. The increased stiffness of the parallel elastic components of muscle proteins, as a result of infiltration of adipose and connective tissue, could be a predominant factor underlying the prolonged contraction time of the twitch in old age (Larsson and Salviati, 1989).

## Muscle Enzyme Changes

The literature examining the question of enzymatic changes during the aging process reveals conflicting results. Although some investigators claim the oxygen-utilizing capacity of muscle cells is well maintained with age (Aniansson et al., 1981), other investigators speculate that there is an age-related decrement in the metabolic and oxidative capacity of skeletal muscle cells. These latter claims have been made in accordance with lower numbers and smaller size of mitochondria (Orlander et al., 1978), and a diminished activity of oxidative enzymes such as adenosinetriphosphatase and succinate dehydrogenase (Ermini, 1976).

Evidence also exits both for and against decreased glycolytic enzyme activities with aging. Aniansson and colleagues (1981) showed that phosphofructokinase (PFK), lactate dehydrogenase (LDH), hexokinase and phosphorylase activities in male and female subjects do not appear to change between the ages of 16 and 83 years. Moreover, Larsson and coworkers (1978) observed the maximal activity of PFK to remain unchanged with age, yet LDH activity decreased significantly in this study. The difference in results of the various studies may be related to physical activity habits of the subjects, however these issues continue to be investigated. In light of the biochemical results, enzymatic changes are not the primary factor responsible for the decline of muscular strength with age.

## Summary

The aging process both within and between individuals varies as genetic, environmental, social, and economic factors play a role. Research has not always been able to isolate the alterations in muscle due solely to biological aging from those resulting

from inactivity or pathology. Therefore, the relative importance of the various age-related changes occurring in the neuromuscular system, to the loss of strength, cannot be determined at this time. Although there is a substantial decrease in muscle strength in most individuals with advancing age, and these decrements in strength are greatly reduced or eliminated when corrected for muscle mass (Frontera et al., 1991). Thus it appears that sarcopenia is the major factor in the age-related decline in muscle strength. In addition to the neuromuscular age-related changes described in the previous section, there are various hormonal changes that may also contribute to the strength loss that occurs as one ages.

## Hormone Status and Human Aging

Another area of research that has acquired significant attention in recent years has been the hormonal changes associated with aging. Biological aging is identified with several other factors such as physical activity, psychological status, nutritional status, disease and the use of medication and drugs. All of these factors may play an important role in the age-related variations of numerous hormones. The study of hormone changes in the aged generally involves the analysis of modifications in the biological rhythms and the parameters characterizing the rhythm (i.e., modifications of the period, shift in the acrophase, increase or decreased amplitude and 24-hour means). One of the rhythms most frequently investigated is the circadian rhythm (24-hour rhythm). An earlier study conducted by Rusak and Zucker (1979) found the circadian rhythm to be centrally generated by a pacemaker located in the suprachiasmatic nuclei of the hypothalamus. Morphological and neurochemical alterations in the suprachiasmatic nuclei have been found in older humans and are possibly responsible for the age-related changes in the 24-hour hormonal rhythms generated by this circadian pacemaker (Weiland and Wise, 1990). Although the circadian rhythm has been shown to persist in healthy elderly subjects, a number of 24-hour hormonal rhythms are dampened and/or advanced in the old adult. The hormones that are of concern in this review are testosterone, GH, cortisol, and estradiol. The age-associated changes in serum levels of SHBG are also important in order to describe the TT and FT levels associated with aging.

## Testosterone

Testosterone is a steroid hormone that is a lipid, synthesized from cholesterol. Although testosterone has several major physiological roles, the regulation of protein synthesis in the target tissue is undoubtedly the principal action. One of the major sites of testosterone action is in skeletal muscle. Testosterone is secreted in circadian rhythms with minimal levels occurring in late evening and maximal levels in the early morning. Testosterone is synthesized in the smooth endoplasmic reticulum of Leydig cells and is controlled via a negative feedback loop (Figure 2-1). Once released into the bloodstream, testosterone is present as both free and bound forms. Approximately 98% of testosterone circulating in the plasma is found in its bound form, the majority attached to the high-infinity, low-capacity protein SHBG, with most of the remaining bound form attached to the low-affinity, high-capacity carrier albumin. The remaining 1-2% of testosterone travels as FT in the blood. It has been speculated that the biologically available testosterone consists of both the FT and the albumin bound testosterone (Pardridge, 1981).

Testosterone progressively decreases after the age of 60 years and the mean testosterone levels at the age of 80 are only about 60% of the mean levels at the age of 20-50 years (Vermeulen and Kaufman, 1995). In the past, the amount of testosterone production in older men has been very controversial. Studies carried out on morning samples showed diminished concentrations in the aged (Vermeulen et al., 1972; Stearns et al., 1974), whereas studies carried out on early afternoon samples revealed normal testosterone concentrations (Harman and Tsitouras, 1980; Sparrow et al., 1980). The discrepancies between these studies are partially related to the decreased circadian amplitude of plasma testosterone levels in older men at different times in the day. Bremner and coworkers (1983) found that elderly men show significantly lower concentrations of plasma testosterone during morning samples (02:00-13:00), although at other times (14:00-01:00) testosterone levels did not differ significantly between the two age groups. The decrease in testosterone levels is also accompanied by an increase in luteinizing hormone levels, thus suggesting a decline in Leydig cell function with age (Kaufman et al., 1991).

## Estradiol, SHBG, and Free Testosterone

In males, the conversion of circulating testosterone through aromatization in peripheral tissue accounts for approximately 50% of the circulating estrogenic hormones. The remainder is derived directly from the testes and the adrenal gland (Longcope et al., 1969). In an early study conducted by Stearns et al. (1974), no significant differences were found in the estradiol concentrations of 282 male subjects aged 18-96. However more recently, investigations have provided evidence which reveals an increase in total plasma estradiol concentrations in aging men (Kley et al., 1974; Rubens et al., 1974; Pirke and Doerr, 1975; Greenblatt et al., 1976; Drafta et al., 1982). This increase is associated with an elevated binding of estrogen to its protein carrier, as well as an increase in free estradiol levels (Rubens et al, 1974; Pirke and Doerr, 1975). Hemsell and coworkers (1974) examined the mechanism behind the elevated estrogen levels during aging in men and found there to be an increased aromatization of androstenedione to estrone by peripheral tissues, thus suggesting that the peripheral conversion of androgen is the source of the elevated estradiol levels in older males.

In a Massachusetts Male Aging Study conducted by Gray et al. (1991), it was found that FT decreases at a rate of 1.2%/year in male subjects between the age of 39-70 years. This decline in the FT levels is more pronounced and occurs earlier age than the reduction found in total testosterone (Vermeulen et al., 1996). The prominent decline of FT is accompanied by an age-related increase of the SHBG capacity (Vermeulen et al., 1971; Gray et al., 1991; Hakkinen and Pakarinen, 1993; Vermeulen et al., 1996). Kley and coworkers (1974) found a high correlation between free estrogen levels and SHBG capacity. This relation makes it seem likely that an increased estradiol production in older men is responsible for the elevation of SHBG and the fall in FT. In addition, the plasma concentration of SHBG in men can be increased by the administration of estrogens (Anderson, 1974). Despite these findings, it has also been found that endogenous estrogens in normal males within the physiological range are poor predictors of SHBG and can explain little of the variation in its concentration (Longcope et al., 1990). In addition, other investigations have also found there to be no significant age effects on the hormonal concentrations of estradiol (Meikle et al., 1982; Ho et al., 1987;

Tenover et al., 1987; Montanini et al., 1988; Vermeulen et al., 1996). The cause of the increase in SHBG is still largely unknown, however it has been postulated that decreased levels of GH and insulin-like growth factor I (IGF-I) levels associated with aging may also be involved (Vermeulin and Kaufman, 1995).

## Growth Hormone

GH is a peptide hormone that is produced by the somatotrophic cells of the anterior pituitary gland and is regulated by growth hormone releasing hormone (Figure 2-2). Growth hormone is an important anabolic hormone with stimulatory effects on protein synthesis and stimulates the liver and other body cells to secrete the growth-promoting peptide hormone IGF-I. GH is also directly associated with organic metabolism of various tissues by increasing circulating concentrations of free fatty acids and inhibiting glucose uptake by peripheral tissues. In a recent study conducted by Winer and coworkers (1990), the 24-hour profile of GH in normal young adults was demonstrated to consist of stable low levels abruptly interrupted by secretory pulses. This study also demonstrated that GH release was closely related to slow wave sleep, with the major secretory episode of GH occurring within minutes of the onset of sleep.

Although GH is normally produced throughout life, there is a progressive decrease of the total daily GH secretion from adolescence to old age (with a particularly rapid decline between 15 and 30 years of age). A recent study has revealed that the GH production rate decreases by 14% and the GH half-life falls by 6% each decade after 20 years of age (Iranmanesh et al., 1991). van Coevorden et al. (1991) found that in normal men over the age of 65, the daily GH secretion is only a third of the amount secreted by younger men aged 20 to 27 years. In addition, van Coevorden and coworkers (1991) also found the daytime levels of plasma GH seem to remain relatively unmodified, however nocturnal GH secretion is reduced with age and is associated with a decrease in the amplitude of the pulses rather than a decrease in the frequency of pulses.

## Cortisol

Cortisol is a steroid hormone that is also derived from cholesterol and one of its primary physiological roles is assisting in the maintenance of blood glucose homeostasis. One of the ways in which cortisol stimulates glucose formation is through amino acid release from muscle (proteolysis). Due to this role in glucose homeostasis and several other effects, cortisol is considered to be a catabolic hormone. Cortisol is synthesized in the adrenal cortex of the adrenal gland and is regulated through the hypothalamic-pituitary-adrenal axis (Figure 2-3). Once secreted into the blood cortisol primarily travels bound to corticosteroid-binding globulin. The circadian pattern of plasma cortisol in healthy young individuals reveal high early morning maximal levels around 7:00 - 8:00 which decline throughout the day with minimal levels centered around midnight followed by an abrupt elevation during late sleep (Weitzman et al., 1971). Sleep-patterns, activity levels and feeding-patterns all have important influences on the cortisol rhythm.

In a meta-analysis of the 24-hour profile of plasma cortisol levels across varying age groups, Van Couter and colleagues (1996) found that the circadian pattern of plasma cortisol is not altered in older subjects. Although this general 24-hour pattern does not change with age, the results of the meta-analysis reveal a progressive and gradual increase in the 24-hour mean levels from the second to the eighth decade. Basal cortisol levels were found to increase by 20 to 50% between 20 and 80 years of age (Van Couter et al., 1996). The overall increase results from an increase in the nadir (minimum level) and a shorter quiescent period (Waltman et al., 1991; Kern et al., 1996; Van Couter et al., 1996). The active free fraction of plasma cortisol is also elevated in the elderly. This increase is thought to be possibly related to a decrease in the concentration of binding proteins or to a decrease in the binding capacity of these proteins with aging (Touitou et al., 1983). In addition, Touitou and coworkers (1983) found no seasonal rhythmicity of cortisol in the elderly whereas such seasonal variations occur in young adult men. In conjunction with the other age-related changes that affect muscle strength, an increase in the catabolic environment may be accountable for further muscle atrophy and augment the decline in strength.

## Summary of Hormonal Changes and Aging

Although the data regarding age-related hormonal changes is still quite scarce, several studies have demonstrated specific changes to the 24-hour hormonal rhythms. The research tends to support the notion that, in aging men, testosterone and GH seem to progressively decrease whereas cortisol, SHBG and estradiol tend to have increased levels. Despite whether the hormone levels increase or decrease, the general secretory patterns are generally maintained, and the alterations in hormone concentrations occur progressively with age. Although the literature has demonstrated that the aging process is associated with hormonal changes, the mechanisms behind these changes are relatively unclear and require further investigation.

## Resistance Training in the Elderly

Strength conditioning is generally defined as "training in which the resistance against which a muscle generates force is progressively increased over time" (Evans, 1992). It has been stated that resistance exercise can delay and possibly even reverse many adverse effects of aging which tend to reduce the physical activity level and the quality of life in the elderly (Lampman, 1987). In a study conducted by Klitgaard and coworkers (1990b), elderly subjects with different training backgrounds (strength-trained, swimmers and runners) were examined for differences in muscle function and morphology. The study revealed that strength trained elderly men were the only group with similar muscle strength and muscle CSA to young sedentary individuals. In contrast, the swimmers and runners had similar muscle strength and CSA to untrained elderly controls. These results suggest that regularly performed strength training, of sufficient intensity, can compensate for age-related muscle wasting. In light of these findings, we can no longer consider strength loss as an inevitable consequence of aging. Therefore, strength training may be the primary mode of exercise for the maintenance of muscular strength and CSA in the elderly.

In order to prescribe safe and effective strength training programs for the elderly, several factors must be considered. The following three variables are generally used to define a fitness program:

- Intensity of training
- Frequency of sessions
- Duration of each session and the training program

Although resistance training studies in the elderly have examined various questions regarding program protocols, more research is necessary to determine how to manipulate these variables to optimize training effects for this age group. Furthermore, health and safety issues are extremely important and one must minister special care when designing exercise programs for the elderly.

## Intensity of Training

The intensity of training seems to be a critical variable for increases in strength (Porter and Vandervoort, 1995). The degree of strength gain attainable in older subjects appears to be dependent upon the intensity of the training stimulus, and it appears that programs employing 70-80% of one repetition maximum (1RM) produces the greatest strength gains in the elderly (Fiatarone and Evans, 1993). Low intensity training studies have produced strength increases of less than 20% (deVries, 1970; Agre et al., 1988), whereas 12 weeks of high intensity training (80% of 1RM) in older adults has produced increases of up to 227% in the 1RM of knee flexion and extension (Frontera et al., 1988). In another study (Fiatarone et al., 1990), strength gains in knee extension of up to 174% was found in 8 weeks of high intensity resistance training (80% of 1RM). These high intensity resistance training studies (80% 1RM) in the elderly (Frontera et al., 1988; Fiatarone et al., 1990; Morganti et al., 1995) also indicate that the strength gains do not plateau throughout the training program. Another study however, using a lower intensity program, found strength gains that tended to plateau (Pyka et al., 1994a). Therefore, it seems that in order for older individuals to make significant gains in strength, a sufficient intensity of 70-80% of 1RM is required.

The intensity of a training session also depends on other variables such as the cadence of the repetitions, and the amount of rest between sets. Maximal strength in the general population is developed by performing multiple sets to fatigue consisting of one to eight repetitions (Stone and Kroll, 1986). The American College of Sports Medicine (1995) advises that elderly individuals should not train with a resistance that is too heavy to complete at least eight repetitions, and the movements should be performed slowly, (concentric phase 2 seconds; eccentric phase 3 seconds) with proper form, through the entire range of motion. Therefore, eight to twelve repetitions to fatigue (momentary failure) per exercise would be classified as high-intensity training for the elderly (Fiatarone from Drought, 1994). Since ninety-five percent of muscle recovery from fatigue occurs in two and a half minutes (Stone and Kroll, 1986), one to three minutes of rest between sets are recommended for rest intervals. Less time is necessary when muscles are not worked to failure (Hurley, 1995).

Another variable to consider when devising a weight program is the training volume (i.e., sets, repetitions, and weight). This variable is very important because Hakkinen and Pakarinen (1994) found that excessive volumes of training may lead to limited increases or even decreases in strength. The number of exercise sets should depend on the number of exercises performed, the muscle groups being trained and practical issues such as session duration (Hurley, 1995). Each exercise should be performed for 2-3 sets as the individual becomes conditioned to tolerate multiple sets (Fiatarone from Drought, 1994).

In a review conducted by Kraemer (1988), it is suggested that three exercise variables may affect hormone concentrations following resistance exercise in young individuals. First, there needs to be an adequate exercise intensity (80% of 1RM) (Guezennec et al., 1986). Secondly, a moderate to high volume (i.e., mass X repetitions X weight) of exercise in each training session is necessary (Weiss et al., 1983). Finally, there must be a large amount of muscle tissue used in the exercise session (Fahey et al., 1976; Guezennec et al., 1986). Furthermore, Kraemer and coworkers (1991) have suggested that exercise protocols utilizing shorter rest periods (<1minute) augments the magnitude of the hormonal response to high-intensity resistance training.

## Frequency of Sessions

The frequency of training is the second variable that determines the type of program. Dedrick and Clarkson (1990) investigated the effects of eccentric exercise in young and older women and found that eccentric actions produce muscle damage from which older subjects recover more slowly. Since resistance training involves both eccentric and concentric actions, approximately 48 hours of recovery time should be allowed between sessions using a particular muscle group for adaptation to occur (Hurley, 1995; Porter and Vandervoort, 1995). Most studies have used a frequency of three sessions per week, however Hicks and coworkers (1991) demonstrated comparable increases in 1RM with only two sessions per week.

## Duration of Each Session and the Training Program

The term duration in a resistance training program refers to two different domains; 1) the duration of the training session and 2) the duration of the training program. Virtually all studies conducted on the elderly implement training sessions that last approximately one hour. It is presumed that there is very little benefit in exercising in the weight room for more than an hour since most gains can be accomplished with workouts lasting approximately one hour. Therefore, the duration of each exercise session should not exceed one hour.

The duration of the training program is another very important variable that researchers must consider when setting up a strength study. It was originally believed that strength gains due to resistance training in the elderly were caused entirely by neurological factors (Moritani and deVries, 1980). However, more recent strength training studies of older adults has shown muscle hypertrophy to occur after as little as eight to twelve weeks of training. (Frontera et al., 1988; Fiatarone et al., 1990; Roman et al., 1993). It is now the notion that at the beginning of a strength training program (i.e., < 8 weeks) the drastic improvement in strength occurs due to enhanced neurological coordination (Sale, 1988). Thereafter, improvements in strength may be the result of both neurological adaptations and an increase in muscle mass, even in the older adult.

Frontera et al. (1988) found that left thigh girth and strength measurements were significantly greater at twelve weeks than at 6 weeks of training. These findings suggest that adaptations to strength training occur for at least 12 weeks. Therefore, an optimal adaptive response in elderly individuals may not be elicited with only six weeks of training. Morganti et al. (1995) demonstrated that individuals improve in strength in a stepwise fashion. Therefore, if this stepwise fashion is representative of a usual physiologic response to strength training in older individuals, previous studies of shorter duration may have continued to show increases if followed for a longer duration. A study conducted by McCartney and colleagues (1995) demonstrate that strength continues to increase over 42 weeks of resistance training, although the greatest gains occurred within the first 12 weeks of training after which the rate slowed down. Therefore, when conducting an exercise study with older adults, a strength training program of twelve weeks in duration seems to elicit significant strength and sufficient physiological adaptation, with an efficient cost and time investment.

## Mechanisms of Strength Gain

Numerous studies have demonstrated that elderly individuals can increase strength through resistance training (Frontera et al., 1988; Fiatarone et al., 1990; Roman et al., 1993; McCartney et al., 1995). It has been only recently that studies have attempted to determine the mechanisms (morphologic, neurologic and intrinsic muscle properties) that contribute to strength gains in older individuals. Yarasheski et al. (1993) determined that the ability of the muscles to hypertrophy is not impaired by aging, and the rate of protein synthesis in response to short-term resistance training is similar in both young and older subjects. Researchers have also analyzed the hypertrophy gains in older adults at the microscopic level, and results from several studies have shown that enlargement of both type I and type II muscle fiber areas occurs in response to strength training (Frontera et al., 1988; Brown et al., 1990; Roman et al., 1993; Pyka et al., 1994a). In addition, the Klitgaard et al. (1990b) study revealed that elderly men with strength training background had a MHC composition (vastus lateralis muscle) that was similar to young sedentary individuals. Both groups exhibited a higher content of MHC

type IIa and type IIb and a correspondingly lower content of MHC type I than elderly swimmers, runners and sedentary controls. These results indicate that habitual strength training may prevent the isoform shift in MHC that occurs during the aging process.

As mentioned previously, a large amount of the increase in strength due to resistance training is attributed to neural adaptations (Sale, 1988). Sale (1988) has also demonstrated that strength increases are not totally transferable to different modes of exercise (i.e., concentric training to eccentric strength or vice versa), therefore a certain amount of neural adaptations (i.e., motor coordination) is gained through performing the specific training actions.

Increases in the rate of force development, and a slowing of relaxation time was reported by Brown and coworkers (1990) after 12 weeks of resistance training in older adults. The authors speculate that this adaptation should allow strength-trained older adults to use lower motor-unit activation rates to achieve maximal force production. Rice and colleagues (1993) found that the intrinsic contractile properties were slowed after both 12 and 24 weeks of resistance training in elderly men. Although the mechanisms responsible for slowed twitch properties remain unknown, the authors speculate that the changes may be due to either excitation-contraction coupling processes or changes in musculo-tendinus compliance.

Hicks and coworkers (1992) suggest that resistance training in older adults may cause possible changes in the excitability of the muscle membrane. The results from this study seem to correspond with the Klitgaard and Clausen (1989) investigation that found increased sites for potassium-sodium transport system in older trained men. These changes in the $Na^{+}$-$K^{+}$ system or muscle membrane excitability could allow peak tension to be achieved more rapidly and possible help prevent falls in the elderly.

## Summary of Resistance Training in the Elderly

Although age-related changes do occur, it appears that even very old individuals can make adaptations in their neuromuscular system if the resistance training program is progressive and of sufficient intensity. Although research has demonstrated that elderly individuals can benefit from a high-intensity resistance training program much like

younger individuals, there appears to be a trade-off between optimal muscle adaptation and safety recommendations for the elderly. When developing a resistance program for older adults, various precautions must be taken into consideration. However, despite these safety hazards, even very old nursing home residents with chronic conditions have been able to successfully complete strength training programs (Fiatarone et al., 1990). Furthermore, the training program must be continued for as long as possible as a 32% loss of maximum strength was observed after only 4 weeks of detraining (Fiatarone et al., 1990).

## Hormonal Adaptations to Resistance Training in the Elderly

Changes in strength and muscle morphology as well as changes in hormone levels occur during the aging process. It has also been known that androgens are known to play an important role in muscle hypertrophy and strength development. Therefore, age-related changes in human muscle strength characteristics could possibly be a consequence of the alterations in hormone balance. Since only a limited number of investigations examining hormonal changes consequent to resistance training in the elderly have been conducted, little experimental information is available on possible inter-relationships between muscle strength characteristics and serum hormonal changes in the elderly.

### Testosterone

Young sedentary males regularly demonstrate significant increases in resting serum testosterone levels as a result of prolonged resistance training (Johnson et al., 1983; Hakkinen et al., 1985; Staron et al., 1994). However in sedentary elderly males, resistance training investigations have not demonstrated this same increase in basal serum testosterone levels. In a study performed by Craig and coworkers (1989), no significant changes were found in basal testosterone levels of elderly males after 12 weeks of resistance training. However, the intensity of training was not reported and if a low intensity program was implemented, the intensity may be a contributing factor for the

lack of significant results. A study conducted by Hakkinen and Pakarinen (1994) also demonstrated no significant changes in serum testosterone concentrations of elderly male and female subjects during a 12-week strength training program. Another study conducted by Ryan et al. (1994) hypothesized that a 16-week strength training program would increase basal testosterone concentrations, however the results obtained in this study also demonstrated no significant changes.

**Estradiol, SHBG, and Free Testosterone**

There is an extreme lack of information regarding the hormonal response of SHBG, FT and estradiol to high-intensity resistance training in elderly male subjects. A study conducted by Hakkinen and Pakarinen (1994) demonstrated that there were no significant changes in both the basal SHBG and FT concentrations in response to a resistance training program. More research in this area is required to confirm these results.

**Growth Hormone**

Resistance exercise is known to be an acute stimulus for GH secretion in healthy young men (Hakkinen et al., 1988; Craig et al., 1989). Craig and coworkers (1989) found no significant changes in GH levels after 12-weeks of strength training in nine elderly male subjects. Although there was substantial improvement in muscular strength over the course of 16 weeks of strength training in a study conducted by Ryan and coworkers (1994), there was no significant changes in the basal serum GH levels in 21 subjects between the ages of 51 and 71. The results obtained in a study performed by Pyka et al. (1994b) also supports the previous results in that 14 elderly subjects between the ages of 64 and 78 years showed no significant change in GH concentrations over the course of a year-long strength training study. The results from these studies therefore provide evidence that resistance training does not produce significant changes in basal GH concentrations in the elderly.

A study in which resistance training was supplemented with hormone therapy revealed that GH treatment, in combination with a resistance training program, did not produce a greater increase in muscle protein synthesis, muscle mass, or strength and no greater decrease in body fat or myofibrillar protein breakdown, than an identical exercise program without GH supplementation (Yarasheski et al., 1995). These results therefore suggest that resistance training may be as effective in producing an anabolic environment in the elderly as GH treatment.

## Cortisol

Many studies analyzing the anabolic hormonal environment associated with resistance training usually measure cortisol, a catabolic hormone as well. Strength studies of young sedentary males produced significant decreases in basal cortisol levels as a result of prolonged resistance training (Hakkinen et al., 1985; Staron et al., 1994), therefore favoring an enhanced anabolic environment. In contrast however, studies using elderly subjects reveal no significant changes in cortisol levels in response to resistance training (Hakkinen and Pakarinen, 1994). Moreover, Pyka and coworkers (1992) demonstrated that subjects over the age of 60 showed no acute change in serum cortisol levels in response to resistance exercise at various exercise intensities.

## Safety of Resistance Training in the Elderly

Although the importance and benefits of muscular strength training in the elderly have recently been studied extensively, the safety of the subjects is of extreme importance. Therefore the benefits to individuals involved in this type of training should be obtained with minimal risk. Various investigators have used 1RM strength testing for assessment of muscular strength and in the prescription of training intensities. However few studies have analyzed the safety of this method in the senior population. In a study conducted by Pollock and colleagues (1991), results indicated that during 1RM strength testing 11/57 subjects incurred an injury. The investigators thus concluded that the elderly are more fragile and may be more susceptible to musculoskeletal injury during

high-intensity, low repetition strength testing. In another study conducted by Shaw and coworkers (1995), it was found that although many subjects complained of muscle soreness (n = 58), only 2 out of 83 subjects resulting in injury due to maximal 1RM strength testing. The authors of this study believe that with proper preparation, the 1RM can be a safe assessment tool for muscular strength in the geriatric population. Although special care must be taken when performing these tests, the abilities of the elderly may be routinely underestimated.

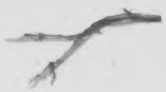
## Adherence and Attendance of Resistance Exercise in the Elderly

Two other important factors that may cause problems during a training study are a lack of adherence and attendance. Adherence refers to whether a subject completes the program and attendance describes the number of sessions participated in by each subject. Pollock et al. (1991) found that in a 26-week strength training program for subjects in their seventies, adherence was 87% and the average attendance was 97.8%. In addition to having a high investigator-to-subject ratio, the authors believed that their higher than average adherence rates were a result of a variety of training times, the ability to conveniently make up missed sessions, the effect of feedback by periodic testing and having sessions conducted in a pre-arranged, safe, well-lit facility.

## Chapter Summary

Exercise is considered one of the most dominant interventions that holds great promise in improving health and promoting independence in the elderly. It is well known that there are age-related changes in muscle strength, function as well as in hormone concentrations, however at this time, little is known about the chronic effects of resistance training on muscle strength and function or of the hormonal status in the older adult. Investigations have revealed that resistance training can increase strength and cross-sectional area of the muscle in elderly subjects if a sufficient training stimulus is provided. However, significant alterations in the hormonal status have not been demonstrated in these resistance training studies. Due to the paucity of experimental

information available on possible inter-relationships between muscle strength characteristics and serum hormonal changes in the elderly, more research is necessary. When implementing a training study, there are many factors that need to be considered. These factors include the form of training, the type of assessments, and any special needs or considerations of the population in question.



## References

American College of Sports Medicine. (1995). Position stand on osteoporosis and exercise. Medicine and Science in Sports and Exercise, 27 (4), i-vii Review.

Agre, J.C., Pierce, L.E., Raab, D.M., McAdams, M., & Smith, E.L. (1988). Light resistance training and stretching exercise in elderly women: effect upon strength. Arch Phys Med Rehabil, 69, 273-276.

Alen, M., Pakarinen, A., Hakkinen, K., & Komi, P.V. (1988). Responses of serum androgenic-anabolic and catabolic hormones to prolonged strength training. International Journal of Sports Medicine, 9 (3), 229-233.

Anderson, D.C. (1974). Sex-hormone binding globulin. Clinical Endocrinology, 3, 69-96.

Aniansson, A., Grimby, G., Hedberg, M., & Krotkiewski, M. (1981). Muscle morphology, enzyme activity and muscle strength in elderly men and women. Clin Physiol, 1, 73-86.

Aniansson, A., Sperling, L., Rundgren, A., & Lehnberg, F. (1983). Muscle function in 75-year-old men and women. Scandinavian Journal of Rehabilitation Medicine, Suppl 9, 92-102.

Booth, F.W., Weeden, S.H., & Tseng, B.S. (1994). Effect of aging on human skeletal muscle and motor function. Medicine and Science in Sports and Exercise, 26 (5), 556-560.

Bremner, W.J., Vitiello, M.V., & Prinz, P.N. (1983). Loss of circadian rhythmicity in blood testosterone levels with aging in normal men. Journal of Clinical Endocrinology and Metabolism, 56 (6), 1278-1281.

Brown, A.B., McCartney, N., & Sale, D.G. (1990). Positive adaptations to weight-lifting training in the elderly. Journal of Applied Physiology, 69 (5), 1725-1733

Clarkson, P.M., Kroll, W., & Melchionda, A.M. (1981). Age, isometric strength, rate of tension development and fiber type composition. Journal of Gerontology, 36, 648-653.

Craig, B.W., Brown, R., & Everhart, J. (1989). Effects of progressive resistance training on growth hormone and testosterone levels in young and elderly subjects. Mechanisms of Ageing and Development, 49, 159-169.

Dedrick, M.E. and Clarkson, P.M. (1990). The effect of eccentric exercise on motor performance in young and older women. European Journal of Applied Physiology, 60, 183-186.

deVries, H.A. (1970). Physiological effects of an exercise training regimen upon men aged 52 to 88. Journal of Gerontology, 25, 325-336.

Drafta, D., Schindler, A.E., Stroe, E., & Neacsu, E. (1982). Age-related changes of plasma steroids in normal adult males. Journal of Steroid Biochemistry, 17, 683-687.

Drought, J.H. (1994). Resistance training and strength benefits for elderly individuals. Strength and Conditioning, June, 26-30.

Ermini, M. (1976). Aging changes in mammalian skeletal muscle. Gerontology, 22, 301-316.

Evans, W.J. (1992). Exercise, Nutrition and Aging. Journal of Nutrition, 122, 796-801.

Fahey, T.D., Rolph, R., Moungmee, P., Nagel, J., & Mortara, S. (1976). Serum testosterone, body composition and strength of young adults. Medicine and Science in Sports and Exercise, 8, 31-34.

Fiatarone, M.A., Marks, E.C., Ryan, N.D., Merideth, C.N., Lipstiz, L.A., & Evans, W.J. (1990). High-intensity strength training in nonagenarians. JAMA, 263 (22), 3029-3034.

Fiatarone, M.A. and Evans, W.J. (1993). The etiology and reversibility of muscle dysfunction in the aged. The Journals of Gerontology, 48 (Special Issue), 77-83.

Frontera, W.R., Meredith, C.N., O'Reilly, K.P., Knuttgen, H.G., & Evans, W.J. (1988). Strength conditioning in older men: Skeletal muscle hypertrophy and improved function. Journal of Applied Physiology, 64 (3), 1038-1044.

Frontera, W.R., Hughes, V.A., Lutz, K.J., & Evans, W.J. (1991). A cross-sectional study of muscle strength and mass in 45- to 78-year-old men and women. Journal of Applied Physiology, 71 (2), 644-650.

Gray, A., Feldman, H.A., McKinlay, J.B., & Longcope, C. (1991). Age, disease, and changing sex hormone levels in middle-aged men: results of the Massachusetts Male Aging Study. Journal of Clinical Endocrinology and Metabolism, 73 (5), 1016-1025.

Greenblatt, R.B., Oettinger, M., & Bohler, C.S.S. (1976). Estrogen-androgen levels in ageing men and women: therapeutic considerations. Journal of the American Geriatrics Society, 24, 173-178.

Greig, C.A., Botella, J., & Young, A. (1993). The quadriceps strength of healthy elderly people after eight years. Muscle and Nerve, 16, 6-10.

Gutman, E. and Hanzlikova, V. (1972). Basic mechanisms of ageing in the neuromuscular system. Mechanisms of Ageing and Development, 1, 327-349.

Guezennec, Y., Leger, L., Lhoste, F., Aymonod, M., & Pesquies, P.C. (1986). Hormone and metabolite response to weight-lifting training sessions. International Journal of Sports Medicine, 7, 100-105.

Hakkinen, K., Pakarinen, A., Alen, M., & Komi, P.V. (1985). Serum hormones during prolonged training of neuromuscular performance. European Journal of Applied Physiology, 53, 287-293.

Hakkinen, K., Pakarinen, A., Alen, M., Kauhanen, H., & Komi, P.V. (1988). Neuromuscular and hormonal responses in elite athletes to two successive strength training sessions in one day. European Journal of Applied Physiology, 57, 133-139.

Hakkinen, K. and Pakarinen, A. (1993). Muscle strength and serum testosterone, cortisol and SHBG concentrations in middle-aged and elderly men and women. Acta Physiol Scan, 148, 199-207.

Hakkinen, K. and Pakarinen, A. (1994). Serum hormones and strength development during strength training in middle-aged and elderly males and females. Acta Physiol Scan, 150, 211-219.

Harman, S.M. and Tsitouras, P.D. (1980). Reproductive hormones in aging men. I. Measurements of sex steroids, basal luteinizing hormone and Leydig cell response to human chorionic gonadotrophin. Journal of Clinical Endocrinology and Metabolism, 51, 35-40.

Hemsell, D.L., Grodin, J.M., Brenner, P.F., Siiteri, P.K., & Macdonald, P.C. (1974). Plasma precursors of estrogen. II. Correlation of extent of conversion of plasma androstenedione to estrone with age. Journal of Clinical Endocrinology and Metabolism, 18, 966-970.

Hicks, A.L., Cupido, C.M., Martin, J., & Dent, J. (1991). Twitch potentiation during fatiguing exercise in the elderly: the effects of training. European Journal of Applied Physiology, 63, 278-281.

Hicks, A.L., Cupido, C.M., Martin, J., & Dent, J. (1992). Muscle excitation in elderly adults: the effects of training. Muscle and Nerve, 15, 87-93.

Ho, K.Y., Evans, W.S., Blizzard, R.M., Veldhuis, J.D., Merriam, G.R., Samojlik, E., Furlanetto, R., Rogol, A.D., Kaiser, D.L., & Thorner, M.O. (1987). Effects of sex and age on the 24-hour profile of growth hormone in man: importance of endogenous estradiol concentrations. Journal of Clinical Endocrinology and Metabolism, 64 (1), 51-58.

Hurley, B. (1995). Strength training in the elderly to enhance health status. Medicine, Exercise, Nutrition and Health, 4, 217-229.

Iranmanesh, A., Lizarralde, G., & Veldhuis, J.D. (1991). Age and relative adiposity are specific negative determinants of the frequency and amplitude of growth hormone (GH) secretory bursts and the half-life of endogenous GH in healthy men. Journal of Clinical Endocrinology and Metabolism, 73, 1081-1088.

Johnson, C.C., Stone, M.H., Byrd, R.J., & Lopez-S, A. (1983). The response of serum lipids and plasma androgens to weight training exercise in sedentary males. Journal of Sports Medicine, 23, 39-44.

Kaufman, J.M., Giri, M., Deslypere, J.P., Thomas, G., & Vermeulen, A. (1991). Influence of age on the responsiveness of the gonadotrophs to luteinizing hormone-releasing hormone in males. Journal of Clinical Endocrinology and Metabolism, 72, 221-224.

Kern, W., Dodt, C., Born, J., & Fehm, H.L. (1996). Changes in cortisol and growth hormone secretion during nocturnal sleep in the course of aging. Journal of Gerontology, Medical Sciences, 51A (1), M3-M9.

Kley, H.K., Nieschlag, E., Bidlingmaier, F., & Kruskemper, H.L. (1974). Possible age dependent influence of estrogens on the binding of testosterone in plasma of adult men. Hormone Metabolism and Research, 6, 213-215.

Klitgaard, H. and Clausen, T. (1989). Increased total concentration of Na-K pumps in vastus lateralis muscle of old trained human subjects. Journal of Applied Physiology, 67 (6), 2491-2494.

Klitgaard, H., Zhou, M., Schiaffino, S., Betto, R., Salviati, G., & Saltin, B. (1990a). Ageing alters the myosin heavy chain composition of single fibres from human skeletal muscle. Acta Physiol Scand, 140, 55-62.

Klitgaard, H., Mantoni, M., Schiaffino, S., Ausoni, S., Gorza, L., Laurent-Winter, C., Schnohr, P., Saltin, B. (1990b). Function, morphology and protein expression of ageing skeletal muscle: a cross-sectional study of men with different training backgrounds. Acta Physiol Scand, 140, 41-54.

Kraemer, W.J. (1988). Endocrine responses to resistance exercise. Medicine and Science in Sports and Exercise, 20 (5), S152-S157.

Kraemer, W.J., Gordon, S.E., Fleck, S.J., Marchitelli, L.J., Mello, R., Dziados, J.E., Friedl, K., Harmen, E., Maresh, C., & Fry, A.C. (1991). Endogenous anabolic hormonal and growth factor responses to heavy resistance exercise in males and females. International Journal of Sports Medicine, 12 (2), 228-235.

Lampman, R.M. (1987). Evaluating and prescribing exercise for elderly patients. Geriatrics, 42 (8), 63-76.

Larsson, L., Sjodin, B., & Karlsson, J. (1978). Histochemical and biochemical changes in human skeletal muscle with age in sedentary males, age 22-65 years. Acta Physiol Scand, 103, 31-39.

Larsson, L., Grimby, G., & Karlsson, J. (1979). Muscle strength and speed of movement in relation to age and muscle morphology. Journal of Applied Physiology, 46 (3), 451-456.

Larsson, L. (1983). Histochemical characteristics of human skeletal muscle during aging. Acta Physiol Scand, 117, 469-471.

Larsson, L. and Salviati, G. (1989). Effects of age on calcium transport activity of sarcoplasmic reticulum in fast- and slow-twitch rat muscle fibers. Journal of Physiology, 419, 253-264.

Lexell, J., Hendriksson-Larsen, K., Winblad, B., & Sjostrom, M. (1983). Distribution of different fiber types in human skeletal muscles: Effects of aging studied in whole muscle cross sections. Muscle and Nerve, 6, 588-595.

Lexell, J., Taylor, C.C., & Sjostrom, M. (1988). What is the cause of the ageing atrophy? Total number, size, and proportion of different fiber types studied in whole vastus lateralis muscle from 15- to 83-year old men. Journal of Neurological Science, 84, 275-294.

Lexell, J. and Downham, D.Y. (1991). The occurrence of fiber type grouping in healthy human muscle: a quantitative study of cross-sections of whole vastus lateralis from men between 15 and 83 years. Acta Neuropathol, 81, 377-381.

Longcope, C., Kato, J., & Horton, R. (1969). Conversion of blood androgens to estrogens in normal adult men and women. Journal of Clinical Investigations, 48, 2191-2195.

Longcope, C., Goldfield, S.R.W., Brambilla, D.J., & McKinlay, J. (1990). Androgens, estrogens, and sex hormone-binding globulin in middle aged men. Journal of Clinical Endocrinology and Metabolism, 71 (6), 1442-1446.

McCartney, N., Hicks, A.L., Martin, J., & Webber, C.E. (1995). Long-term resistance training in the elderly: Effects on dynamic strength, exercise capacity, muscle, and bone. Journal of Gerontology, Biological Sciences, 50A (2), B97-B104.

Meikle, A.W., Stanish, W.M., Taylor, N., Edwards, C.Q., & Bishop, C.T. (1982). Familial effects on plasma sex-steroid content in man: testosterone, estradiol and sex-hormone-binding globulin. Metabolism, 31 (1), 6-9.

Montanini, V., Simoni, M., Chiossi, G., Baraghini, G.F., Velardo, A., Baraldi, E., & Marrama, P. (1988). Age-related changes in plasma dehydroepiandrosterone sulphate, cortisol, testosterone, and free testosterone circadian rhythms in adult men. Hormone Research, 19, 1-6.

Morganti, C.M., Nelson, M.E., Fiatarone, M.A., Dallal, G.E., Economos, C.D., Crawford, B.M., & Evans, W.J. (1995). Strength improvements with 1 year of progressive resistance training in older women. Medicine and Science in Sports and Exercise, 27 (6), 906-912.

Moritani, T. and deVries, H.A. (1980). Potential for gross muscle hypertrophy in older men. Journal of Gerontology, 35 (5), 672-282.

Nelson, R.M., Soderberg, G.L., & Urbscheit, N.L. (1984). Alteration of motor-unit discharge characteristics in aged humans. Physical Therapy, 64, 29-34.

Orlander, J., Keissling, K., Larsson, L., & Karlsson, J. (1978). Skeletal muscle metabolism and ultrastructure in relation to age in sedentary men. Acta Physiol Scand, 104, 249-261.

Pardridge, W.M. (1981). Transport of protein-bound hormones into tissues *in Vivo*. Endocrine Reviews, 2 (1), 103-123.

Pirke, K.M. and Doerr, P. (1975). Age-related changes in free plasma testosterone, dihydrotestosterone, and oestradiol. Acta Endocrinology, 80, 171-178.

Pollock, M.L., Carrol, J.F., Graves, J.E., Leggett, S.H., Braith, R.W., Limicher, M., & Hagberg, J.M. (1991). Injuries and adherence to walk/jog and resistance training programs in the elderly. Medicine and Science in Sports and Exercise, 23 (10), 1194-1200.

Porter, M.M. and Vandervoort, A.A. (1995). High-intensity strength training for the older adult - A review. Topics in Geriatric Rehabilitation, 10 (3), 61-74.

Pyka, G., Wiswell, R.A., & Marcus, R. (1992). Age-dependent effect of resistance exercise on growth hormone secretion in people. Journal of Clinical Endocrinology and Metabolism, 75, 404-407.

Pyka, G., Lindenberger, E., Charette, S., & Marcus, R. (1994a). Muscle strength and fiber adaptations to a year-long resistance training program in elderly men and women. Journal of Gerontology, Medical Sciences, 49 (1), M22-M27.

Pyka, G., Taaffe, D.R., & Marcus, R. (1994b). Effect of sustained program of resistance training on the acute growth hormone response to resistance exercise in older adults. Hormone Metabolism and Research, 26, 330-333.

Rice, C.L., Cunningham, D.A., Paterson, D.H., & Dickinson, J.R. (1993). Strength training alters contractile properties of the triceps brachii in men aged 65-78 years. European Journal of Applied Physiology, 66, 275-280.

Roman, W.J., Fleckenstein, J., Stray-Gundersen, J., Alway, S.E., Peshock, R., & Gonyea, W.J. (1993). Adaptations in the elbow flexors of elderly males after heavy-resistance training. Journal of Applied Physiology, 74 (2), 750-754.

Rubens, R., Dhont, M., & Vermuelen, A. (1974). Further studies on Leydig cell function in old age. Journal of Clinical Endocrinology and Metabolism, 39, 40-45.

Rusak, B. and Zucker, I. (1979). Neural regulation of circadian rhythms. Physiol Rev, 59, 449-526.

Ryan, A.S., Treuth, M.S., Rubin, M.A., Miller, J.P., Nicklas, B.J., Landis, D.M., Pratley, R.E., Libanati, C.R., Gundberg, C.M., & Hurley, B.F. (1994). Effects of strength training on bone mineral density: hormonal and bone turnover relationships. Journal of Applied Physiology, 77 (4), 1678-1684.

Sale, D.G. (1988). Neural adaptation to resistance training. Medicine and Science in Sport and Exercise, 20, S135-S145.

Sato, T., Akatsuka, H., Kuniyoshi, K., & Tokoro, Y. (1984). Age changes in size and number of muscle fibers in human minor pectoral muscle. Mechanisms of Aging and Development, 28, 99-100.

Shaw, C.E., McCully, K.K., & Posner, J.D. (1995). Injuries during the one repetition maximum assessment in the elderly. Journal of Cardiopulmonary Rehabilitation, 15, 283-287.

Sparrow, D., Bone, R., & Rowe, J.W. (1980). The influence of age, alcohol consumption and body build on gonadal function in men. Journal of Clinical Endocrinology and Metabolism, 51, 508-512.

Staron, R.S., Karapondo, D.L., Kraemer, W.J., Fry, A.C., Gordon, S.E., Falkel, J.E., Hagerman, F.C., & Hikida, R.S. (1994). Skeletal muscle adaptations during early phase of heavy-resistance training in men and women. Journal of Applied Physiology, 76 (3), 1247-1255.

Stearns, E.L., MacDonnell, J.A., Kaufman, B.J., Padau, R., Lucman, T.S., Winter, J.S.D., & Faiman, C. (1974). Declining testicular function with age, hormonal and clinical correlates. American Journal of Medicine, 57, 761-766.

Stone, W.J. and Kroll, W.A. (1986). Sports Conditioning and Strength Training. Programs for Athletic Competition. Allyn and Bacon, Inc.

Tenover, J.S., Matsumoto, A.M., Plymate, S.R., & Bremner, W.J. (1987). The effects of aging in normal men on bioavailable testosterone and luteinizing hormone secretion: responses to clomophene. Journal of Clinical Endocrinology and Metabolism, 65 (6), 1118-1126.

Tomlinson, B.E. and Irving, D. (1977). The numbers of limb motor neurons in the human lumbrosacral cord throughout life. Journal of Neurological Sciences, 34, 213-219.

Tomonaga, M. (1977). Histochemical and ultrastructural changes in senile human skeletal muscle. J Am Geriatr Soc, 3, 125-131.

Touitou, Y., Sulon, J., Bogdan, A., Reinberg, A., Sodoyez, J.C., & Demey-Ponsart, E. (1983). Adrenocortical hormones, ageing and mental conditions: Seasonal and circadian rhythms of plasma 18-hydroxy-11-deoxycorticosterone, total and free cortisol and urinary corticosteroids. Journal of Endocrinology, 96, 53-64.

van Coevorden, A., Mockel, J., Laurent, E., Kerkhofs, M., L'Hermite-Baleriaux, M., Decoster, C., Neve, P., & Van Couter, E. (1991). Neuroendocrine rhythms and sleep in aging men. American Journal of Physiology, 260, E651-E661.

Van Couter, E., Leproult, R., & Kupfer, D.J. (1996). Effects of gender and age on the levels and circadian rhythmicity of plasma cortisol. Journal of Clinical Endocrinology and Metabolism, 81 (7), 2468-2473.

Vermeulen, A., Stoica, T., & Verdonck, L. (1971). The apparent free testosterone concentration, an index of androgenicity. Journal of Clinical Endocrinology and Metabolism, 33, 759-767.

Vermeulen, A., Ruben, R., & Verdonck, L. (1972). Testosterone secretion and metabolism in male senescence. Journal of Clinical Endocrinology and Metabolism, 34, 730-735.

Vermeulen, A. and Kaufman, J.M. (1995). Ageing of the Hypothalamo-Pituitary-Testicular Axis in men. Hormone Research, 43, 25-28.

Vermeulen, A., Kaufman, J.M., & Giagulli, V.A. (1996). Influence of some biological indexes on sex hormone-binding globulin and androgen levels in aging or obese males. Journal of Clinical Endocrinology and Metabolism, 81 (5), 1821-1826.

Waltman, C., Blackman, M.R., Chrousos, G.P., Riemann, C., & Marman, S.M. (1991). Spontaneous and glucocorticoid-inhibited adrenocorticotropic hormone and cortisol secretion are similar in healthy young and old men. Journal of Clinical Endocrinology and Metabolism, 73, (3): 495-502.

Weiland, N. and Wise, P. (1990). Aging progressively decreases the densities and alters the diurnal rhythms of alpha-1 adrenergic receptors in selected hypothalamic regions. Endocrinology, 126, 2392-2397.

Weiss, L.W., Cureton, K.J., & Thompson, F.N. (1983). Comparison of serum testosterone and androstenedione responses to weight lifting in men and women. European Journal of Applied Physiology, 50, 413-419.

Weitzman, E., Fukushima, D., Nogeire, C., Roffwarg, H., Gallagher, T., & Hellman, L. (1971). Twenty-four hour pattern of the episodic secretion of cortisol in normal subjects. Journal of Clinical Endocrinology and Metabolism, 33, 14-22.

Winer, L.M., Shaw, M.A., & Bauman, C. (1990). Basal plasma growth hormone levels in man: new evidence for rhythmicity of growth hormone secretion. Journal of Clinical Endocrinology and Metabolism, 70, 1678-1686.

Yarasheski, K.E., Zachwieja, J.J., & Bier, D.M. (1993). Acute effects of resistance exercise on muscle protein synthesis rate in young and elderly men and women. American Journal of Physiology, 265, E210-E214.

Yarasheski, K.E., Zachwieja, J.J., Campbell, J.A., & Bier, D.M. (1995). Effect of growth hormone and resistance exercise on muscle growth and strength in older men. American Journal of Physiology, 31, E268-E276.

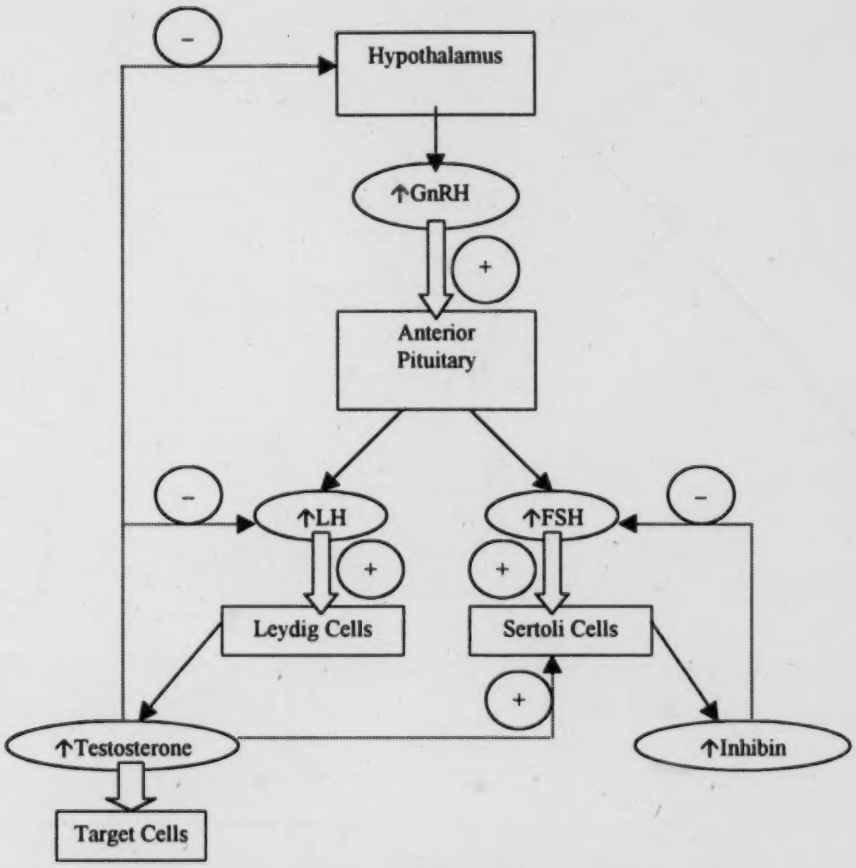



Figure 2-1. Interactions of the physiological regulation of the hypothalamic-pituitary-gonadal axis. The (-) denotes that the input is inhibited. The (+) denoting stimulation.

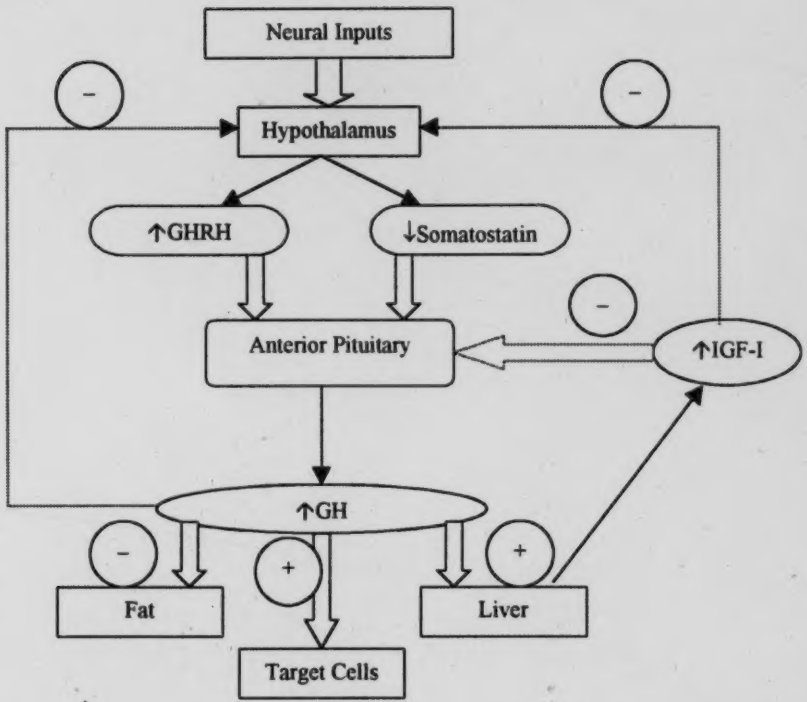



Figure 2-2. Interactions of the physiological regulation of GH. The (-) denotes that the input is inhibited. The (+) denoting stimulation.

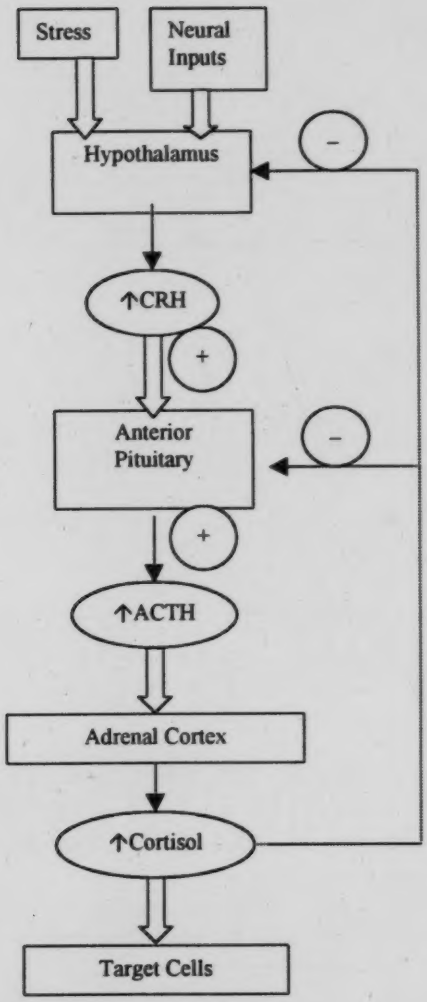



Figure 2-3. Interactions of the physiological regulation of the CRH-ACTH-cortisol sequence. The (-) denotes that the input is inhibited. The (+) denoting stimulation.

# CHAPTER 3

# THE EFFECT OF HIGH-INTENSITY RESISTANCE TRAINING ON THE HORMONAL AND STRENGTH RESPONSES IN HEALTHY ELDERLY MALES

## Introduction

The growing number of individuals over the age of 65 years has resulted in extensive research of the elderly and the aging process. It is well known that the decline in muscular strength is associated with increasing age and it is particularly prevalent at the onset of the sixth decade. A number of changes, such as a decline in muscle fiber number (Sato et al., 1984; Lexell et al., 1988), decreased muscle CSA (Lexell et al., 1988), decline in motor unit number (Lexell and Downham, 1991), abnormalities at the neuromuscular junction (Gutman and Hanzlikova, 1972; Tomonaga, 1977), deterioration of the excitation-contraction coupling process (Larsson and Salviati, 1989), and enzymatic changes (Larsson et al., 1978; Aniansson et al., 1981), have been linked with aging and recognized as potential explanations for the diminished strength in the aged. The possible mechanisms for these changes include disuse atrophy (Sallis et al., 1985; Rickli and Busch, 1986); cumulative diseases (Tinetti, Williams & Mayewski, 1986); malnutrition (Dastur et al., 1982; Lewis et al., 1986); and hormonal changes (Vermeulin and Kaufman, 1995; Van Couter et al., 1996; Iranmanesh et al., 1991).

Research has focussed on examining specific means to retard or possibly even reverse these changes in order to maintain physical function and independence for the elderly population. Moreover, to circumvent sarcopenia, the maintenance and improvement of the anabolic environment in the muscle is essential. Studies investigating testosterone and human growth hormone administration in the elderly demonstrate an increase in skeletal muscle strength and protein synthesis (Urban et al., 1995; Rudman et al., 1990). However, hormone therapy may cause potential side effects such as impaired glucose tolerance, hypertension and fluid retention, (Marcus et al., 1990; Salomon et al., 1989). In young healthy males, high-intensity resistance exercise

has shown to enhance the basal testosterone concentrations (Hakkinen et al., 1085; Busso et al., 1990) and human growth hormone release after resistance exercise (Vanhelder et al., 19984; Craig and Kang, 1994). However there is a paucity of information regarding the effect of high-intensity resistance exercise on hormonal responses in elderly males. Therefore, the purpose of this study was to examine the effects of a 16-week high-intensity resistance training program on basal hormonal concentrations of total testosterone, free testosterone, human growth hormone, estradiol, urinary cortisol and sex hormone-binding globulin in elderly men. In addition, this study examined the effects of resistance training on strength, lean body mass, girth measures, percent body fat, hemoglobin and hematocrit. It was hypothesized that there would be significant changes in muscular strength, percent body fat, lean body mass and girth measures, combined with significant changes in potentially anabolic hormones (total testosterone, free testosterone, human growth hormone), estradiol, sex hormone-binding globulin and urinary cortisol levels.

## Methods

### Subjects

Twenty-two healthy elderly men, ages 62 to 76, from the metropolitan Edmonton area volunteered for the study, which was approved by the ethics board of the Faculty of Physical Education and Recreation at the University of Alberta. One subject sustained an injury not related with the study and another subject could not fulfil the time commitment of the training, resulting in 20 individuals completing the study. All subjects were active in daily activities and had participated in regular aerobic exercise at least once a week prior to the study. However, none had participated in a regular resistance-training program within the previous 6 months. Subjects passed a medical examination for participation in a resistance training program, were free of cardiac and orthopedic disease, were not on any medication, passed a participant readiness questionnaire and signed an informed consent after the purpose and procedures of the study were explained (Appendix A). See Table 3-1 for subject characteristics.

## Experimental Design and Training

After baseline assessments, the subjects were matched for strength and randomly assigned to a control group (CT) (n = 10) or to a resistance training group (RT) (n = 10). The CT was asked to continue with habitual activity patterns and requested to not participate in any formal fitness training programs until the study was completed. Verbal communication with the CT group and the RT group was used to assure that their physical activity patterns and daily diets did not change during the study period. The RT was required to complete a 16-week, periodized, resistance-training program. The strength training program consisted of four, 4-week cycles with a mean intensity of 70.2% (range 61-80%) of 1RM. Within the cycles, exercise intensities ranged from 50-90% of 1RM. The subjects performed 3-5 sets of 3 to 12 repetitions with approximately 3 minutes of rest between sets. The training was performed three times a week (Monday, Wednesday, and Friday) under direct supervision to ensure proper technique and decrease the possible risk of injury. Exercise sessions were preceded by a 5-10 minutes warm-up that included either walking, stationary biking or rowing followed by light stretching. The exercises included bench press, dumbbell arm curls, triceps extensions, lat pull-downs, shoulder press, standing calf raises, inclined leg press and leg extensions. All exercises were performed on Apex and Cybex weight training equipment or free weights. For the RT group, muscular strength was re-evaluated on the last training day of the cycle every four weeks to account for any neurological or morphological changes that could affect strength, and the training programs were adjusted accordingly to maintain the proper intensity of training throughout the study. The CT group was tested for strength at baseline and at the end of 16 weeks. The resistance training programs were produced using a computerized strength program called Strength Disk II (B.E. SOFTWARE, Lincoln, Nebraska).

## Assessment of Maximal Muscular Strength

Prior to assessment of maximal muscular strength, all subjects were required to attend a one-hour familiarization session. During this time the subjects were given

advice regarding proper lifting techniques and permitted to practice the technique of the exercises being tested. The leg press exercise was performed on an inverted incline leg press that was set at an angle of 45 degrees. The range of motion for the leg press exercise required lowering the weight to a 90-degree knee joint bend, followed by lifting the weight back up to full knee extension. All other exercises were tested throughout a full range of motion in a slow and controlled fashion. Strength was assessed as a voluntary one repetition maximum (1RM) for bench press and leg press and a multiple repetition maximum (mRM) for all other exercises (dumbbell arm curls, triceps extensions, lat pull-downs, shoulder press, standing calf raises and seated leg extensions). The 1RM test measures the heaviest weight that can be lifted once, throughout a complete range of motion, while adhering to proper technique. The 1RM protocol requires each subject to perform a light warm-up followed by successive sets (5-8 sets) of 1 repetition with progressively heavier weight until the maximal amount of weight that only can be lifted once is determined. The mRM measures the maximal amount of weight that can be lifted for several repetitions utilizing proper technique. The mRM protocol consisted of 2-4 successive sets of 6 to 12 repetitions of progressively heavier weight until volitional failure to perform a further repetition in the range of 6 to 12 repetitions occurred. Approximately three minutes of rest were allowed between all sets to minimize fatigue. The 1RM and mRM tests were repeated within a one-week period and the greatest value of the two scores was used as the baseline value. The strength testing sessions began with either the 1RM bench press or leg press followed by all other strength exercises in random order.

**Analytical Methods**

After 4 hours of fasting, resting blood samples (10ml) were taken by a registered nurse from an antecubital vein. The blood samples were drawn between 16:00 and 18:00, two days prior to the beginning of training and approximately 48 hours after the exercise sessions at the end of weeks 8 and 16. Serum samples for the hormonal analysis of total testosterone (TT), free testosterone (FT), estradiol, human growth hormone (GH), sex hormone-binding globulin (SHBG) were kept frozen at -80 °C until assayed. The assays

of serum TT and FT were performed by radioimmuno assays (RIA) using reagent kits from INCSTAR Corporation (Stillwater, Minnesota - USA), estradiol and GH kits supplied by Sorin Biomedica Diagnostics (Saluggia, Vercelli - Italy) and SHBG kits supplied by EURO/DPC Ltd. (Llanberis, Gwynedd - United Kingdom).

Subjects collected a urine sample over a 24-hour period in a sterile plastic bottle prior to the beginning of training and 36-48 hours after the exercise sessions at the end of weeks 8 and 16. The urine collection started with the first void upon getting up for the day and was terminated on the last void prior to getting up on the second day. Total volume of the urine sample was measured and 10-ml samples for the hormonal analysis of cortisol were kept frozen at -80 °C until assayed. The analysis of the 24-hour urinary cortisol was performed with a RIA from INCSTAR Corporation (Stillwater, Minnesota - USA), according to the manufacturer's procedures for unextracted urinary cortisol.

All of the subjects samples and control values were analyzed in duplicate according to the procedures outlined by the manufacturer. The intra-assay coefficient of variance was determined by meaning the values of all the duplicates and any duplicates above 20 % were reanalyzed. The intra-assay coefficient of variances of the sample duplicates were TT (5.0%), FT (4.65%), GH (4.66%), estradiol (6.93%), cortisol (7.41%), and SHBG (5.68%). Due to re-analyses and a delay in receiving the GH RIA kit, there were two freeze-refreeze actions on the samples prior to performing the GH assay. Whole blood samples were used to determine Hb levels and these were kept refrigerated at 4 °C, in the dark, for 24-48 hours. Hb was determined using the cyanmethemoglobin technique as described by Tietz, 1987. Hct was determined within minutes after the blood was drawn using the microcentrifuge method as described by Tietz, 1987.

A Professional Fitness and Lifestyle Consultant took pre- and post-training anthropometric measurements. Girth measurements included relaxed and tensed mid-arm, mid-leg and chest girths that were taken with a Lufkin anthropometric measuring tape as described by MacDougall, Wenger and Green (1991). Six skinfold measurements were taken using Harpenden skinfold calipers and were recorded as the mean of duplicate or triplicate measurements if a difference of greater than 4 mm between the first two measurements existed. The skinfold sites included subscapular, tricep, iliac crest, chest,

abdominal and mid-thigh. Percent body fat was calculated from the sum of six skinfolds according to Yuhasz (1966). Lean body mass (LBM) was calculated by subtracting fat weight from body weight. In addition, all subjects had their height and weight recorded.

## Statistical Analysis

Standard descriptive statistical methods were used to calculate means and standard deviations. A two-way (group and time) repeated analysis of variance (ANOVA) was used to determine potential differences in anthropometric measures and strength between groups, and before and after training. A two-way ANOVA with repeated measures was also used to determine differences between groups and before and after 8 and 16 weeks of training for all hormone and hematological measurements. A one-way ANOVA with repeated measures was used to determine differences after 4, 8, 12 and 16 weeks of training for bench press and leg press 1 RM. A Newman-Keuls multiple comparison procedure was used to assess any main and/or interaction effects revealed by the ANOVA's. The alpha level was set a priori at $p < 0.05$. All of the statistical analyses were accomplished with the Statistica software package (STATSOFT, Oklahoma City, OK).

# Results

## General Characteristics

The participants in the RT group were not significantly different from the participants in the CT group. Subjects in the RT group attended 97.2% of the scheduled supervised training sessions. There were no training-related injuries. The RT group significantly decreased their percent body fat during the 16-week resistance training (Table 3-2) while there was no changes in the control group. There were no significant changes in any other anthropometric measures in either group.

## Muscular Strength

Initially there were no differences in the maximal strength measures between the groups at baseline with the exception of the left arm dumbbell curl that was significantly different between the CT and the RT. The leg press 1RM significantly increased from 284.85 kg (±48.27) to 366.92 kg (±47.17) during the 16-week strength training program in the RT group (Table 3-3). Figure 3-1 shows the increase in the 1RM of the leg press during the course of the resistance-training period. There was also a significant increase in the bench press 1RM from 59.32 kg (±11.05) to 68.86 kg (±10.61) in the RT group (Table 3-3). Figure 3-2 shows the changes in the bench press 1 RM during the time course of the resistance-training program. There were no significant increases in the strength measures of these exercises in the CT group during this period. The RT group demonstrated significant strength increases in all of the supplementary exercises of the training program, while the CT group did not show any significant changes (Table 3-3).

## Serum and Urinary Hormone Levels

There were no differences between groups in hormonal levels and hematology measurements prior to the training program with the exception of a significant difference in TT between the CT and RT groups (2.94 ±1.01 vs. 4.94 ±1.34 ng/ml), respectively (Table 3-4). There was a significant decrease in TT after training in the RT (Table 3-4). No other significant differences were found in any hormones in either group in response to resistance training. One subject in the CT group had unusually high urinary cortisol levels. Removing him from the data set resulted in a significant main effect for group, with the RT group being higher than the CT group, with levels of 59.60 (±17.82) and 45.49 (±12.18) µg cortisol per 24 hours, respectively. There was no significant difference found in Hct. However, there was a significantly higher Hb level in the RT compared to the CT group after 16 weeks of training (Table 3-4).

## Discussion

It was hypothesized that high-intensity resistance training in elderly males would produce significant changes in strength, lean body mass, girth measures and in particular anabolic and catabolic hormone levels. The findings of this study support the hypothesis that resistance training produces significant changes in strength levels and percent body fat, as there was a significant increase in muscular strength and percent body fat decreased significantly. Furthermore, the results partially support the hypothesis that resistance training produces a significant change in anabolic hormone levels, as there was a significant decrease in TT concentration after resistance training. However the hypotheses for the remaining variables were not supported, as there were no changes in lean body mass, girths or basal hormonal concentrations of FT, GH, estradiol, SHBG and basal urinary cortisol levels in response to the resistance training.

In the present study, changes in the size of the muscle were measured indirectly through the means of LBM and contracted and relaxed girth measures, but no significant changes in these measurements were observed. The no change in LBM of this study can be partially explained by the decline in percent body fat accompanied by a small decrease in body weight, and the absence of change in girth measures. It would be expected that a decrease in percent body fat would be associated with an increase in LBM. However, since the decrease in percent body fat was accompanied with a decrease (non-significant) in body weight, there was no significant change in the LBM of the subjects in this study. Previous studies measuring girths and LBM offer conflicting results. Some studies reveal increases in a variety of girth measures as a result of resistance training (Taaffe et al., 1994; Sipila and Suominen, 1995; Yarasheski et al., 1995), whereas other investigations have shown no changes (Staron et al., 1994; Ades at al., 1996). Despite research that has shown resistance training to produce muscular hypertrophy in upper (Brown et al., 1990) and lower body (Frontera et al., 1988) muscles, there was no significant change in girth measurements of the upper and lower extremities in the present study. The lack of significant changes in the girth measures provides further support that there was no change in the LBM of the subjects in this study. However, the main problem associated with girth measurements is that changes of the specific components (i.e., muscle, fat or

bone) within the measure cannot be separated. Therefore, any possible increases in muscle size could have been masked by the decrease in body fat. Unless direct measurement of muscle hypertrophy such as muscle biopsies or computer tomography was used, no concrete conclusions can be made regarding possible changes in muscle size. Regardless, the gains in muscular strength must be accounted for and since there were no increases in muscle size or LBM, as indicated by the measurements used in the present study, the adaptations in strength could be possibly attributed to changes in neural mechanisms, such as an increase in motor unit recruitment or an increase in the synchronization of motor units.

One of the goals of this investigation was to determine possible strength changes associated with the training program. The two main strength measures were the leg press and the bench press exercises, while all the other strength exercises were performed to supplement the training program. The time course for the leg press exercise demonstrates that there were significant increases in strength from baseline to 8 weeks. As well, throughout the remaining 8 weeks of the study, strength continued to increase but a further significant increase was not observed until week 16. The time course of strength gains in the leg press exercise agrees with previous research that observed the most notable increases during the first six to eight weeks of a resistance training program (Pyka et al., 1994a; McCartney et al., 1995; Morganti et al., 1995). According to previous research by Sale (1988), the early, rapid increases in strength observed in approximately 4 weeks may be attributed to neural adaptations, while later increases with longer term training (> 4 weeks) may be attributed to skeletal muscle hypertrophy. Bench press strength followed a different time course of adaptations as no significant increases in the bench press 1RM strength occurred until week 12. However, there was a gradual, but not significant, increase in bench press 1RM throughout the 16 weeks of resistance training. Regardless of the time course of adaptations, the resistance-training program in the present study was successful in promoting strength increases in upper and lower body strength of elderly subjects.

The present study found that high-intensity resistance training had little effect on the basal concentrations of FT, GH, estradiol, SHBG, and cortisol. These results are consistent with previous studies of elderly males that also found no significant difference

in basal hormonal changes in FT (Hakkinen and Pakarinen, 1994), GH (Pyka et al., 1992; Pyka et al., 1994b; Ryan et al., 1994), SHBG (Pyka et al., 1992; Hakkinen and Pakarinen, 1994; Ryan et al, 1994; Pyka et al., 1994b) or cortisol (Hakkinen et al., 1985; Pyka et al., 1992; Hakkinen and Pakarinen, 1994; Staron et al., 1994) after resistance training, despite significant increases in strength. The present study also found that TT significantly decreased with resistance training, whereas other studies have reported that TT does not change as a result of resistance training (Craig et al., 1989; Hakkinen and Pakarinen, 1994; Ryan et al, 1994). Research investigating the effect of resistance training on hormone levels, especially TT, have observed either an increase (Johnson et al., 1983; Hakkinen et al., 1985; Staron et al., 1994) or no significant changes in TT levels (Alen et al., 1988; Hickson et al., 1994) in young sedentary males. Moreover, a significant decrease in TT levels in young sedentary men, in response to resistance training, has not been previously reported in past research. Conversely, it is interesting to note that GH, the other anabolic hormone analyzed, had a relative increase of 22.7%. Although, in agreement with previous research investigating GH and resistance exercise (Pyka et al., 1992; Pyka et al., 1994b; Ryan et al., 1994), the change was not statistically significant. The results of this study seem to suggest that elderly men can increase strength with resistance training despite little change in basal anabolic hormone levels and even a decrease in TT levels.

The decreases in the TT concentrations are difficult to explain but may be explained in different ways. First, testosterone production (gonadal or extragonadal) may be decreased or there may have been an increase in the hepatic clearance rate resulting in diminished basal serum TT concentration. Alternatively, there may have been an increase in extra-hepatic clearance through an increase in target cell utilization. It has been suggested that bound testosterone serves as a buffer and storage for the free fraction and protects the hormone from rapid catabolism (Daughaday, 1959). Testosterone is bound to both the high-affinity, low capacity protein SHBG, and the low-affinity, high-capacity carrier albumin (Dunn et al., 1981). However, it is the SHBG concentration that is the major determinant in the amount of testosterone that is biologically accessible. A widely held hypothesis in endocrinology is that serum FT is the exclusive marker for biologically available testosterone. However, other researchers believe that the half-

dissociation time of testosterone from albumin is fast (<1 second), and within the capillary transit time through testosterone target tissue, therefore allowing the albumin-bound testosterone to also be biologically accessible (Pardridge, 1981; Cumming and Wall, 1985). The review conducted by Pardridge (1981) attempts to advance the concept that the large protein-bound moiety of plasma testosterone may also be available for transport into tissues. If this theory is correct, even though the SHBG levels did not change (thus indicating no change in testosterone binding), then the decrease in TT found in the present study may even be attributed to an increase in extra-hepatic clearance of the SHBG-bound testosterone via target tissue utilization. However, the suggestion of an increase in target tissue utilization should have resulted in an increase in cross-sectional area of the muscle. Alternatively, the relative (and significant) decrease in the TT levels was 17.2% and was accompanied by a non-significant decline in the FT with a relative decrease of 14.5%. By looking at these relative values, the decrease in TT may be partially explained through decreases in the FT levels, however whether the decreases in TT and FT were a result of increased extra-hepatic clearance or hepatic clearance rates cannot be determined at this time.

The decrease in TT levels could have resulted from a decrease in production (gonadal or extragonadal), an increase in hepatic clearance of the testosterone hormone, an increased extra-hepatic target tissue utilization, or even a combination of these. However, the exact mechanism behind the decrease in TT can not be determined at this time. The decrease in TT found in this study requires further research to determine the possible mechanisms. Furthermore, since there was no change in the Hct levels, it can be assumed that the hormonal responses in this study were not affected by fluid shifts in the blood. Although a decrease in TT levels does not help to maintain the anabolic environment in the muscle, potential benefits of diminished TT levels (i.e., prevalence of prostate cancer) in elderly males could be important and should be investigated.

The effect of resistance training in elderly men on estradiol levels has not been adequately investigated. Previous research has found that the increase in SHBG with age seems to correspond to a rise in estradiol concentrations (Kley, 1974). Another aim of this study was to determine whether resistance training could modify the SHBG levels indirectly by lowering the estradiol levels. If estradiol could be decreased through

resistance training, and in turn cause a decrease in SHBG levels, then the FT concentrations could possibly be influenced. The relative estradiol levels of the RT group declined 15.4%, however this was not statistically significant. Although the training tended to reveal a decreasing trend in estradiol concentrations, resistance training did not result in significant changes in concentrations of FT, SHBG or estradiol. Therefore, the results of the present study suggest that resistance training was unable to alter basal FT levels directly or indirectly via estradiol.

Finally, this study found a significant difference between groups in Hb levels post-training. Although there was not a time effect within groups, the RT group had significantly higher Hb levels than the CT group at the end of the 16 weeks. In a resistance training study conducted by Frontera et al. (1990), the Hb values tended to decrease during the 12 weeks of resistance training, however the change did not reach statistical significance (P=0.079). Whether these Hb changes could have reached significance if the Frontera et al. (1990) study was extended to 16 weeks is questionable. The difference between Hb levels between the RT and CT groups in the present study suggests that resistance training in elderly men may promote changes in Hb levels.

## Conclusions

The results of this investigation demonstrate that healthy elderly males can participate in training programs of highly intense resistance activity without injury and with a high level of compliance. The present findings show that older men are capable of significantly increasing maximal upper and lower body strength and significantly decreasing percent body fat in response to high-intensity resistance training. However, these changes were observed despite no change in LBM, body girths or a variety of serum hormone levels. Furthermore, a significant decrease in basal TT concentration was observed after resistance training. Thus, a 16-week, high-intensity resistance-training program does not appear to be an effective means of altering the hormonal status of the elderly men in the present investigation. It is concluded that the resistance-training program followed in the present study was not an effective method of increasing the anabolic environment (i.e., GH and TT) of skeletal muscle in healthy elderly males.

## References


Ades, P.A., Ballor, D.L., Ashikaga, T., Utton, J.L., & Nair, K.S. (1996). Weight training improves walking endurance in healthy elderly persons. Annals of Internal Medicine, 124 (6), 568-572.

Alen, M., Parakinen, A., Hakkinen, K., & Komi, P.V. (1988). Responses of serum androgenic-anabolic and catabolic hormones to prolonged strength training. International Journal of Sports Medicine, 9 (3), 229-233.

Aniansson, A., Grimby, G., Hedberg, M., & Krotkiewski, M. (1981). Muscle morphology, enzyme activity and muscle strength in elderly men and women. Clin Physiol, 1, 73-86.

Brown, A.B., McCartney, N., & Sale, D.G. (1990). Positive adaptations to weight-lifting training in the elderly. Journal of Applied Physiology, 69, (5), 1725-1733.

Busso, T., Hakkinen, K., Pakarinen, A., Carasso, C., Lacour, J.R., Komi, P.V., & Kaukanen, H. (1990). A systems model of training responses in elite weight lifters. European Journal of Applied Physiology, 61, 48-54.

Craig, B.W., Brown, R., & Everhart, J. (1989). Effects of progressive resistance training on growth hormone and testosterone levels in young and elderly subjects. Mechanisms of Ageing and Development, 49, 159-169.

Craig, B.W. and Kang, H. (1994). Growth hormone release following single versus multiple sets of back squats: Total work versus power. Journal of Strength and Conditioning Research, 8 (4), 270-275.

Cumming, D.C. and Wall, S.R. (1985). Non-sex hormone-binding globulin-bound testosterone as a marker for hyperandrogenism. Journal of Clinical Endocrinology and Metabolism, 61 (5), 873-876.

Dastur, D., Manghani, D., Osuntokun, B., Sonrander, P., & Kondo, K. (1982). Neuromuscular and related changes in malnutrition: A review. Journal of Neurol Sci, 55, 207-230.

Daughaday, W.H. (1959). Steroid protein interactions. Physiological Reviews, 39, 885-902.

Dunn, J.F., Nisula, B.C. & Rodbard, D. (1981). Transport of steroid hormones: Binding of 21 endogenous steroids to both testosterone-binding globulin and corticosteroid-binding globulin in human plasma. Journal of Clinical Endocrinology and Metabolism, 53, 58-68.

Frontera, W.R., Meredith, C.N., O'Reilly, K.P., Knuttgen, H.G., & Evans, W.J. (1988). Strength conditioning in older men: Skeletal muscle hypertrophy and improved function. Journal of Applied Physiology, 64 (3), 1038-1044.

Frontera, W.R., Merideth, C.N., O'Reilly, K.P., & Evans, W.J. (1990). Strength training determinants of $VO_{2max}$ in older men. Journal of Applied Physiology, 68 (1), 329-333.

Gutman, E. and Hanzlikova, V. (1972). Basic mechanisms of ageing in the neuromuscular system. Mechanisms of Ageing and Development, 1, 327-349.

Hakkinen, K., Pakarinen, A., Alen, M., & Komi, P.V. (1985). Serum hormones during prolonged training of neuromuscular performance. European Journal of Applied Physiology, 53, 287-293.

Hakkinen, K. and Pakarinen, A. (1993). Muscle strength and serum testosterone, cortisol and SHBG concentrations in middle-aged and elderly men and women. Acta Physiol Scan, 148, 199-207.

Hakkinen, K. and Pakarinen, A. (1994). Serum hormones and strength development during strength training in middle-aged and elderly males and females. Acta Physiol Scan, 150, 211-219.

Hickson, R.C., Hidaka, K., Foster, C., Falduto, M.T., & Chatterton, R.T. (1994). Successive time courses of strength development and steroid hormone responses to heavy-resistance training. Journal of Applied Physiology, 76 (2), 663-670.

Johnson, C.C., Stone, M.H., Byrd, R.J., & Lopez-S, A. (1983). The response of serum lipids and plasma androgens to weight training exercise in sedentary males. Journal of Sports Medicine, 23, 39-44.

Iranmanesh, A., Lizarralde, G., & Veldhuis, J.D. (1991). Age and relative adiposity are specific negative determinants of the frequency and amplitude of growth hormone (GH) secretory bursts and the half-life of endogenous GH in healthy men. Journal of Clinical Endocrinology and Metabolism, 73, 1081-1088.

Kley, H.K., Nieschlag, E., Bidlingmaier, F., & Kruskemper, H.L. (1974). Possible age dependent influence of estrogens on the binding of testosterone in plasma of adult men. Hormone Metabolism and Research, 6, 213-215.

Larsson, L., Sjodin, B., & Karlsson, J. (1978). Histochemical and biochemical changes in human skeletal muscle with age in sedentary males, age 22-65 years. Acta Physiol Scand, 103, 31-39.

Larsson, L. and Salviati, G. (1989). Effects of age on calcium transport activity of sarcoplasmic reticulum in fast- and slow-twitch rat muscle fibers. Journal of Physiology, 419, 253-264.

Lewis, M., Sieck, G., Fournier, M., & Belman, M. (1986). Effect of nutritional deprivation on diaphragm contractility and muscle fiber size. Journal of Applied Physiology, 60, 596-603.

Lexell, J., Taylor, C.C., & Sjostrom, M. (1988). What is the cause of the ageing atrophy? Total number, size, and proportion of different fiber types studied in whole vastus lateralis muscle from 15- to 83-year old men. Journal of Neurological Science, 84, 275-294.

Lexell, J. and Downham, D.Y. (1991). The occurrence of fibre type grouping in healthy human muscle: a quantitative study of cross-sections of whole vastus lateralis from men between 15 and 83 years. Acta Neuropathol, 81, 377-381.

MacDougall, J.D., Wenger, H.A., & Green, H.J. (1991). Physiological Testing of the High-Performance Athlete. Second Edition. Human Kinetics Books, Champaign, Illinois.

Marcus, R., Butterfield, G., Holloway, L., Gilliland, L., Baylink, D.J., Hintz, R.L., & Sherman, B.M. (1990). Effects of short term administration of recombinant human growth hormone to elderly people. Journal of Clinical Endocrinology and Metabolism, 70 (2), 519-527.

McCartney, N., Hicks, A.L., Martin, J., & Webber, C.E. (1995). Long-term resistance training in the elderly: Effects on dynamic strength, exercise capacity, muscle, and bone. Journal of Gerontology: Biological Sciences, 50A (2), B97-B104.

Morganti, C.M., Nelson, M.E., Fiatarone, M.A., Dallal, G.E., Economos, C.D., Crawford, B.M., & Evans, W.J. (1995). Strength improvements with 1 year of progressive resistance training in older women. Medicine and Science in Sports and Exercise, 27 (6), 906-912.

Pardridge, W.M. (1981). Transport of protein-bound hormones into tissues *in Vivo*. Endocrine Reviews, 2 (1), 103-123.

Pyka, G., Wiswell, R.A., & Marcus, R. (1992). Age-dependent effect of resistance exercise on growth hormone secretion in people. Journal of Clinical Endocrinology and Metabolism, 75, 404-407.

Pyka, G., Lindenberger, E., Charette, S., & Marcus, R. (1994a). Muscle strength and fiber adaptations to a year-long resistance training program in elderly men and women. Journal of Gerontology, Medical Sciences, 49 (1), M22-M27.

Pyka, G., Taaffe, D.R., & Marcus, R. (1994b). Effect of sustained program of resistance training on the acute growth hormone response to resistance exercise in older adults. Hormone and Metabolism Research, 26, 330-333.

Rudman, D., Feller, A.G., Nagraj, H.S., Gergans, G.A., Lalitha, P.Y., Goldberg, A.F., Schlenker, R.A., Cohn, L., Rudman, I.W., & Mattson, D.E. (1990). Effects of human growth hormone in men over 60 years old. The New England Journal of Medicine, 323, 1-6.

Rickli, R. and Busch, S. (1986). Motor performance of women as a function of age and physical activity level. Journal of Gerontology, 41, 645-649.

Ryan, A.S., Treuth, M.S., Rubin, M.A., Miller, J.P., Nicklas, B.J., Landis, D.M., Pratley, R.E., Libanati, C.R., Gundberg, C.M., & Hurley, B.F. (1994). Effects of strength training on bone mineral density: hormonal and bone turnover relationships. Journal of Applied Physiology, 77 (4), 1678-1684.

Sale, D.G. (1988). Neural adaptation to resistance training. Medicine and Science in Sport and Exercise, 20, S135-S145.

Sallis, J., Haskell, W., Wood, P., & Fortman, S. (1985). Physical activity assessment methodology in the Five City project. American Journal of Epidemiology, 121, 91-106.

Sato, T., Akatsuka, H., Kuniyoshi, K., & Tokoro, Y. (1984). Age changes in size and number of muscle fibers in human minor pectoral muscle. Mechanisms of Aging and Development, 28, 99-100.

Salomon, F., Cuneo, R.C., Hesp, R., & Sonksen, P.H. (1989). The effects of treatment with recombinant human growth hormone on body composition and metabolism in adults with human growth deficiency. New England Journal of Medicine, 321, 1797-1803.

Sipila, S. and Suominen, H. (1995). Effects of strength and endurance training on thigh and leg muscle mass and composition in elderly women. Journal of Applied Physiology, 78 (1), 334-340.

Staron, R.S., Karapondo, D.L., Kraemer, W.J., Fry, A.C., Gordon, S.E., Falkel, J.E., Hagerman, F.C., & Hikida, R.S. (1994). Skeletal muscle adaptations during early phase of heavy-resistance training in men and women. Journal of Applied Physiology, 76 (3), 1247-1255.

Taaffe, D.R., Pruitt, L., Reim, J., Hintz, R.L., Butterfield, G., Hoffman, A.R., & Marcus, R. (1994). Effect of recombinant human growth hormone on the muscle strength response to resistance exercise in elderly men. Journal of Clinical Endocrinology and Metabolism, 79 (5), 1361-1366.

Teitz, N.W. (1987). Fundamentals of Clinical Chemistry. Third Edition. W.B. Sauders Company.

Tinetti, M.E., Williams, T.F., & Mayewski, R. (1986). Fall risk index for elderly patients based on number of chronic disabilities. American Journal of Medicine, 80, 429-434.

Tomonaga, M. (1977). Histochemical and ultrastructural changes in senile human skeletal muscle. Journal of the American Geriatrics Society, 3, 125-131.

Urban, R.J., Bodenburg, Y.H., Gilkison, C., Foxworth, J., Coggan, A.R., Wolfe, R.R., & Ferrando, A. (1995). Testosterone administration to elderly men increases skeletal muscle strength and protein synthesis. American Journal of Physiology, 269, E820-E826.

Van Couter, E., Leproult, R., & Kupfer, D.J. (1996). Effects of gender and age on the levels and circadian rhythmicity of plasma cortisol. Journal of Clinical Endocrinology and Metabolism, 81 (7), 2468-2473.

Vanhelder, W.P., Radomski, M.W., & Goode, R.C. (1984). Growth hormone responses during intermittent weight lifting exercise in men. European Journal of Applied Physiology, 53, 31-34.

Vermeulen, A. and Kaufman, J.M. (1995). Ageing of the Hypothalamo-Pituitary-Testicular Axis in men. Hormone Research, 43, 25-28.

Yarasheski, K.E., Zachweija, J.J., Campbell, J.A., & Bier, D.M. (1995). Effect of growth hormone and resistance exercise on muscle growth and strength in older men. American Journal of Physiology, 31, E268-E276.

Yuhasz, M.S. (1966). Physical Fitness and Sports Appraisal Laboratory Manual. London: The University of Western Ontario.

**Table 3-1.** Subject characteristics of the experimental groups prior to training.

| Group | Age (yr) | Height (cm) | Weight (kg) | Percent Body Fat (%) |
|---|---|---|---|---|
| CT (n=10) | 67.7 ±3.7 | 169.7 ± 8.4 | 84.2 ±11.0 | 18.2 ±3.4 |
| RT (n=10) | 68.3 ±3.2 | 175.5 ±3.5 | 85.0 ±11.6 | 17.0 ±4.2 |

Values are means ± SD. CT = Control Group and RT = Resistance Trained Group. % Body Fat was determined in 10 CT and 9 RT.

**Table 3-2.** Body Composition in older men before and after either 16 weeks of resistance training (RT) or no training (CT).

| | CT (n=10) | | RT (n=10) | |
|---|---|---|---|---|
| Measure | Initial | Final | Initial | Final |
| Body Weight (kg) | 84.2 ±11 | 84.3 ±11.1 | 85.0 ±11.6 | 83.5 ±10.6 |
| Body Fat (%) | 18.2 ±3.4 | †18.2 ±2.7 | 17.0 ±4.2 | *†15.4 ±3.6 |
| LBM (kg) | 68.7 ±7.6 | 68.8 ±7.7 | 69.4 ±8.1 | 69.5 ±7.4 |
| Relaxed Biceps Girth (cm) | 32.1 ±2.4 | 31.2 ±2.8 | 30.9 ±3.2 | 31.2 ±2.7 |
| Flexed Biceps Girth (cm) | 33.1 ±2.4 | 32.6 ±2.6 | 32.4 ±3.0 | 32.9 ±2.8 |
| Relaxed Thigh Girth (cm) | 50.9 ±3.7 | 52.1 ±3.3 | 50.2 ±3.1 | 50.8 ±2.9 |
| Flexed Thigh Girth (cm) | 51.4 ±3.8 | 52.5 ±3.7 | 50.6 ±3.1 | 51.4 ±2.8 |
| Relaxed Chest Girth (cm) | 105.1 ±6.6 | 105.8 ±5.7 | 102.9 ±9.0 | 101.4 ±7.3 |
| Flexed Chest Girth (cm) | 108.5 ±6.0 | 107.8 ±5.7 | 104.3 ±9.3 | 104.2 ±7.2 |

Values are means ± SD. % Body Fat and girth measurements were determined in 10 CT and 9 RT subjects. * Different from initial values ($P < 0.05$); † Difference between the groups after training ($P < 0.05$).

**Table 3-3.** Strength changes in older men before and after either 16 weeks of resistance training (RT) or no training (CT).

| | CT (n=10) | | RT (n=10) | |
|---|---|---|---|---|
| Exercise | Initial | Final | Initial | Final |
| Leg Press (kg) | 290.91 ±58.78 | † 289.75 ±52.54 | 284.85 ±48.27 | *† 366.92 ±47.17 |
| Bench Press (kg) | 60.07 ±8.96 | † 60.98 ±12.48 | 59.31 ±11.05 | *† 68.86 ±10.61 |
| Left Arm Dumbbell Curl (kg) | ¥ 10.06 ±1.89 | † 10.17 ±1.99 | ¥ 11.27 ±1.85 | *† 12.7 ±2.3 |
| Right Arm Dumbbell Curl (kg) | 10.71 ±2.00 | † 11.04 ±1.89 | 11.00 ±1.88 | *† 13.32 ±2.44 |
| Lat Pulldowns (# of 10 lbs. Plates) | 12.50 ±1.52 | † 12.50 ±1.22 | 13.67 ±2.00 | *† 16.00 ±1.50 |
| Triceps Extensions (# of 5 lbs. Plates) | 12.38 ±2.33 | † 12.00 ±1.69 | 12.20 ±1.99 | *† 14.00 ±2.58 |
| Calf Raises (# of 25 lbs. Plates) | 17.63 ±3.02 | † 20.00 ±4.04 | 18.60 ±3.72 | *† 26.90 ±5.04 |
| Leg Extensions (# of 10 lbs. Plates) | 13.22 ±1.92 | † 12.11 ±2.37 | 14.40 ±1.84 | *† 19.70 ±3.53 |
| Shoulder Press (# of 10 lbs. Plates) | 6.43 ±1.40 | † 6.71 ±1.25 | 7.67 ±1.22 | *† 10.00 ±1.22 |

Bench Press statistics were determined in 6 CT and 10 RT subjects. Leg Press statistics were determined in 9 CT and 9 RT subjects. Values are means ± SD. * Different from initial values ($P < 0.05$); † Difference between the groups after training ($P < 0.05$). ¥ Difference between groups at baseline ($P < 0.05$).

**Table 3-4.** Hormone levels in older men before and after either 16 weeks of resistance training (RT) or no training (CT).

| | | CT (n=10) | | | RT (n=10) | |
|---|---|---|---|---|---|---|
| Measure | Initial | Mid | Final | Initial | Mid | Final |
| Testosterone (ng/ml) | ¥ 2.94 ±1.01 | 3.39 ±1.39 | 3.09 ±1.24 | ¥ 4.94 ±1.34 | * 4.24 ±1.33 | * 4.09 ±1.02 |
| Free Testosterone (pg/ml) | 11.11 ±3.35 | 12.12 ±4.26 | 11.91 ±4.88 | 12.96 ±4.82 | 13.19 ±6.48 | 11.08 ±3.87 |
| Cortisol (µg/24hr) | 71.41 ±69.78 | 72.03 ±110.60 | 84.40 ±110.79 | 62.18 ±12.43 | 56.46 ±16.28 | 60.14 ±24.74 |
| Estradiol (pg/ml) | 24.31 ±7.73 | 30.32 ±13.52 | 27.87 ±11.30 | 28.29 ±10.26 | 26.07 ±6.40 | 23.94 ±8.29 |
| SHBG (nmol/L) | 57.44 ±20.36 | 56.11 ±15.31 | 55.94 ±18.95 | 76.98 ±25.68 | 77.95 ±25.67 | 75.06 ±27.05 |
| Growth Hormone (ng/ml) | 1.87 ±2.19 | 0.82 ±1.15 | 0.45 ±0.52 | 2.16± 2.54 | 2.11 ±2.02 | 2.65 ±3.44 |
| Hematocrit (%) | 43.9 ± 3.0 | 44.6 ±2.9 | 43.9 ± 3.4 | 45.0 ± 3.0 | 45.1 ±3.1 | 44.5 ± 2.4 |
| Hemoglobin (g/100 ml) | 14.22 ±1.32 | 15.10 ±1.39 | † 13.58 ±1.03 | 14.88 ±1.50 | 14.88 ±2.16 | † 15.48 ±1.29 |

Values are means ± SD. * Different from initial values ($P < 0.05$); † Difference between the groups after training ($P < 0.05$). ¥ Difference between groups at baseline ($P < 0.05$).

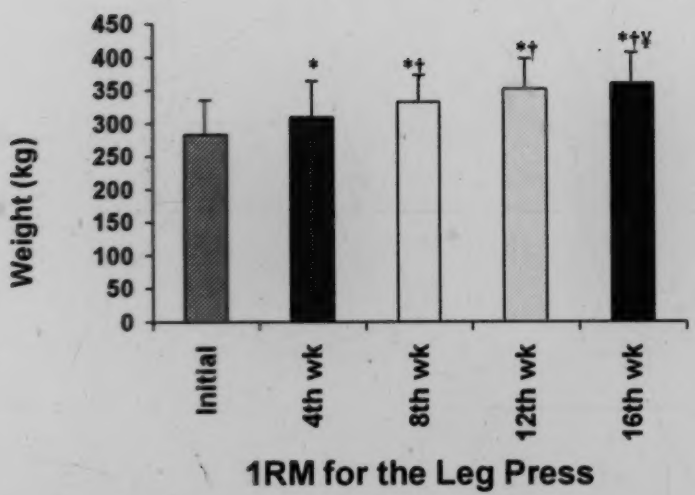



**Figure 3-1.** The changes in the strength measures of the inverted, inclined leg press changes associated with 16 weeks of high-intensity resistance-training in older men. * Different from pre-training measurements ($P < 0.05$). † Different from week 4 ($P < 0.05$). ¥ Different from week 8 ($P < 0.05$).

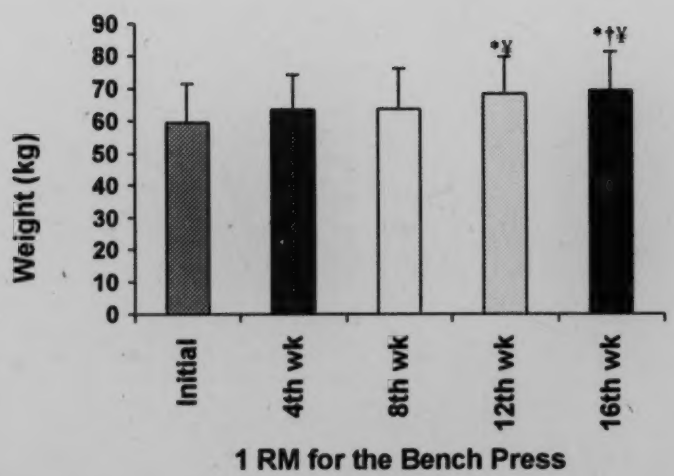



**Figure 3-2.** The changes in strength measures of the bench press exercise associated with 16 weeks of high-intensity resistance-training in older men. * Different from pre-training measurements ($P < 0.05$). † Different from week 4 ($P < 0.05$). ¥ Different from week 8 ($P < 0.05$).

# CHAPTER 4

## GENERAL DISCUSSIONS AND CONCLUSIONS

With the rising age of the population, many scientists are trying to answer the endless questions regarding the aging process. Some scientists believe that aging is not an inevitable process, while others are concerned with maintaining the functional capabilities that are necessary for an individual to remain an integral part of society into old age. Whether the primary goal of the research is to develop anti-aging medical practices, clone mammals for human transplant, or the study of the andropause and sarcopenia, the common goal of all of these investigations is to increase the longevity individuals while maintaining a healthy and functional existence.

This investigation had two main purposes; **1)** to determine whether a 16-week high-intensity resistance training program produces statistically different basal hormonal concentrations in total testosterone, free testosterone, growth hormone, estradiol, cortisol, and the hormone carrier protein sex hormone-binding globulin in elderly men; and **2)** to determine whether the training program produces statistically significant changes in upper and lower body muscular strength. Other variables that were investigated included percent body fat, lean body mass, contracted and relaxed chest, mid-thigh, and mid-biceps measures, hemoglobin and hematocrit.

### Hypotheses

The hypotheses for the different questions, reported respectively, include:

**1)** There would be significant differences between pre- and post-training dependent measurements of basal plasma concentrations of total testosterone, free testosterone, growth hormone, estradiol, sex hormone-binding globulin and basal urinary cortisol concentrations.

**2)** There would be significant differences between pre- and post-training dependent measurements in upper and lower body muscular strength.

## Experimental Design

**1)** Baseline analysis of the strength and basal hormone levels of sedentary elderly male volunteers was performed.
**2)** Subjects were matched for strength and randomly assignment to either a RT or CT group
**3)** Administration of a 16-week, high-intensity, periodized, resistance-training program to the RT group, while preserving the habitual physical activity levels of the CT group.
**4)** Calculation of the main and interaction effects between groups and over time (pre-, mid, and post-training)

Based on the study design, several assumptions were made in order to form the conclusions of this investigation.
**1)** All subjects complied with the proper nutritional and exercise instructions prior to all blood and urine sampling.
**2)** All subjects performed all of the strength tests with maximal physical and mental best efforts.
**3)** Monthly evaluation of strength resulted in accurately prescribed training intensity.
**4)** Control group complied with maintaining habitual physical activity patterns.
**5)** External stresses (such as illness) did not contribute to intra-subject variability.
**6)** Diet and hydration were similar throughout the study for each subject.
**7)** Time of day did not play a role in strength performances.

## General Discussion

According to this study and other research, resistance training does not effectively alter the basal hormonal profile in elderly males (Craig et al., 1989; Hakkinen and Pakarinen, 1994; Ryan et al., 1994; Yarasheski et al, 1995). However, it is important to note that there were various research design problems in these previous studies, such as the intensity and volume of the training, in addition to the exercise protocols, that were not entirely suitable to elicit the hormonal responses observed in the studies involving

younger subjects (Johnson et al., 1983; Hakkinen et al., 1985; Cumming et al., 1987; Alen et al., 1988; Staron et al., 1994). The aim of this study was to correct the experimental design problems and investigate any possible changes in the results. In regards to the hormonal responses of FT, GH, cortisol, estradiol, and SHBG, the hypothesis was not supported and agreed with the current research that has observed no significant changes in the hormonal profile after resistance training in elderly men. However, the present results pertaining to the TT concentrations contradict previous research and support the hypothesis as 16 weeks of high-intensity resistance training produced a statistically significant decrease in basal TT concentrations. Since the actual mechanism behind this response was not investigated, it can only be speculated how this response was induced. Possible mechanisms include decreased production rates (gonadal or extragonadal), increased hepatic clearance rates or increased extra-hepatic clearance. Further research is necessary to determine whether the decrease in TT concentrations can be duplicated and if so, what are the mechanisms behind this decline.

The decrease in TT is also opposite to the testosterone response found in younger male subjects (Johnson et al., 1983; Hakkinen et al., 1985; Staron et al., 1994). This is particularly interesting as the results suggest that, at some point in the aging process, the testosterone response to resistance training may be reversed. Therefore, if these results are reliable, future research needs to investigate the age at which these changes occur as well as the consequences of lower testosterone concentrations. Does the reversal coincide with the age-related decreases in testosterone? What are the potential benefits and/or side effects of impaired testosterone levels? Although the subjects in the RT group experienced a decrease in their basal TT concentrations, these individuals were still able to increase their muscular strength. The time course of the testosterone response demonstrates that the most significant decline in the TT levels occurred during the first 8 weeks of training. This corresponds to the greatest gains in muscular strength. Therefore, from these observations, we may speculate that the decrease in TT levels did not impair the ability to increase strength in the elderly men of the present study.

Another important finding in this investigation was the decrease in percent body fat of the RT group in response to the training. This decrease is supported by several other investigations analyzing the effects of resistance training on body composition in

the elderly (Craig et al., 1989; Hakkinen and Parakinen, 1994; Ryan et al., 1994; Sipila and Suominen, 1995). Therefore, in addition to the benefits associated with increases in strength, individuals can also decrease body fat with resistance training, and both of these benefits may be functionally advantageous to the elderly. The capacity to perform certain actions enhances the opportunity for an independent life. With more strength and less fat mass, movements such as chair rising and stair climbing can be accomplished with less effort. In addition, it has been found that some of the factors that attribute to falls in the elderly are impairment in leg strength, impairment in arm strength, impairment in gait, and impairment in balance (Tinetti et al., 1988; Campbell et al., 1989). It is therefore reasonable to believe that maintaining or increasing strength to an older age may delay these impairments, hence, decreasing the rate of falling in the elderly. By lowering the incidence of falls in the elderly via resistance training, the yearly costs for acute health care, associated with fall-related injuries, should also be reduced.

**Limitations of the Study**

**1)** Since the subjects were volunteers, the selection was not random and the subjects may not be a fully adequate representation of the entire population of healthy males between 62 and 76 years of age.

**2)** Due to the cost of RIA kits, the analysis of hormones were performed in duplicate only.

**3)** Due to difficulties ordering the GH RIA kits, there were two freeze-refreeze actions on all the serum samples prior to running the GH assay.

**4)** Due to the extremely low GH levels identified in particular samples, the accuracy of the values below 0.5ng/ml may be questionable since they do not fall within the proper limits of the standard curve.

## Conclusions

The present investigation has provided evidence that indicates that high-intensity resistance training does not produce any significant changes in basal hormone levels in FT, GH, estradiol, cortisol and SHBG. It seems that even with the significant decrease in TT concentration, upper and lower body strength increased significantly after training. Thus, a 16-week, high-intensity, resistance training program, implemented in elderly individuals can increase total body strength. However, with the exception of a decrease in total testosterone, the training does not appear to be an effective means of significantly altering the hormonal status of these individuals.

## References


Alen, M., Pakarinen, A., Hakkinen, K., & Komi, P.V. (1988). Responses of serum androgenic-anabolic and catabolic hormones to prolonged strength training. International Journal of Sports Medicine, 9 (3), 229-233.

Campbell, A.J., Borrie, M.J., & Spears, G.F. (1989). Risk factors for falls in a community-based prospective study of people 70 years and older. Journal of Gerontology, 44, M112-M117.

Cumming, D.C., Wall, S.R., Galbraith, M.A., & Belcastro, A.N. (1987). Reproductive hormonal responses to resistance exercise. Medicine and Science in Sports and Exercise, 19, 234-238.

Craig, B.W., Brown, R., & Everhart, J. (1989). Effects of progressive resistance training on growth hormone and testosterone levels in young and elderly subjects. Mechanisms of Ageing and Development, 49, 159-169.

Hakkinen, K., Pakarinen, A., Alen, M., & Komi, P.V. (1985). Serum hormones during prolonged training of neuromuscular performance. European Journal of Applied Physiology, 53, 287-293.

Hakkinen, K. and Pakarinen, A. (1994). Serum hormones and strength development during strength training in middle-aged and elderly males and females. Acta Physiol Scan, 150, 211-219.

Johnson, C.C., Stone, M.H., Byrd, R.J., & Lopez-S, A. (1983). The response of serum lipids and plasma androgens to weight training exercise in sedentary males. Journal of Sports Medicine, 23, 39-44.

Ryan, A.S., Treuth, M.S., Rubin, M.A., Miller, J.P., Nicklas, B.J., Landis, D.M., Pratley, R.E., Libanati, C.R., Gundberg, C.M., & Hurley, B.F. (1994). Effects of strength training on bone mineral density: hormonal and bone turnover relationships. Journal of Applied Physiology, 77 (4), 1678-1684.

Sipila, S. and Suominen, H. (1995). Effects of strength and endurance training on thigh and leg muscle mass and composition in elderly women. Journal of Applied Physiology, 78 (1), 334-340.

Staron, R.S., Karapondo, D.L., Kraemer, W.J., Fry, A.C., Gordon, S.E., Falkel, J.E., Hagerman, F.C., & Hikida, R.S. (1994). Skeletal muscle adaptations during early phase of heavy-resistance training in men and women. Journal of Applied Physiology, 76 (3), 1247-1255.

Tinetti, M.E., Speechly, M., & Ginter, S.F. (1988). Risk factors for falls among elderly persons living in a community. New England Journal of Medicine, 319, 1701-1707.

Yarasheski, K.E., Zachweija, J.J., Campbell, J.A., & Bier, D.M. (1995). Effect of growth hormone and resistance exercise on muscle growth and strength in older men. American Journal of Physiology, 31, E268-E276.

# APPENDIX A

- **Informed consent approved by the Faculty of Physical Education and Recreation Ethics Review Committee**

**FACULTY OF PHYSICAL EDUCATION AND RECREATION**
**UNIVERSITY OF ALBERTA**
**EDMONTON, ALBERTA**
**T6G 2H9**

## THE EFFECT OF RESISTANCE TRAINING ON THE HORMONAL RESPONSE IN THE ELDERLY

**Investigator:**
Michael J.E. Souster, BKin 425-5522

## INFORMED CONSENT FOR EXERCISE TESTING AND TRAINING

I,_____(please print your name) agree to participate in a research project conducted by the above named investigators studying the effects of a 16-week high intensity resistance training program on the anabolic hormonal status in elderly men. I agree to participate in the exercise testing and training procedures to the best of my ability. I understand that I may decline to enter, or withdraw from the experiment at any time, for any reason, without any consequences at my own free will. I also understand that the staff conducting the test will discontinue any procedures if any indications of abnormal responses become apparent. I understand that prior to performing any test listed below I will have the opportunity to question and discuss the exact procedures to be followed.

**Physiological Assessments**

**Anthropometry** – measurements of height, weight, arm and leg girths will be recorded.

**1 RM strength tests** – to assess strength levels throughout the course of the study, 1 RM tests will be performed prior to the beginning of week 1, and repeated at the ends of weeks 4, 8, 12 and 16. Done on both the bench press and the leg press, the 1 RM test will be used to determine the maximal amount of weight that each participant can lift once with proper technique.

**Multiple repetitions maximum strength tests (mRM)** – 8 RM tests will be performed prior to the beginning of week one and repeated at the end of weeks 4, 8, 12 and 16 to determine the maximal amount of weight each participant can lift for 6-12 repetitions utilizing proper technique. The exercises for the mRM tests will include: leg extension, leg curl, calf raises, arm curl, tricep extension, lat pull down, and shoulder press.

**Resting blood samples** – 10 ml of blood will be taken by a certified phlebotomist or registered nurse from an antecubital vein. The blood samples will be taken prior to the beginning of week 1 and at the ends of weeks 4, 8, 12, and 16. The subjects will be asked to be in a fasted state for approximately 4 hours prior to the blood sampling.

**24 Hour urine sample** – Subjects will be required to collect their urine for a period of 24 hours prior to the beginning of week 1 and at the end of weeks 4, 8, 12 and 16. A sterile plastic bottle will be supplied to each subject to take home.

**Physiological Training:**

The subjects will be randomly assigned into a control group (n=15) and a resistance training group (n=15). The control group will be required to continue with their daily activities and it will be requested that they abstain from participation in any formal fitness training until the study is completed. The resistance training group will be required to complete a 16 week periodized program. The strength program will begin at a mean intensity of approximately 60%

of 1 RM and progress to 80% of 1 RM. The repetitions will range from 5 to 10 and 2 to 6 sets. The training will be 3 times a week (one and a half hours). The exercises will include bench press, arm curls, tricep extension, lat pull downs, shoulder press, leg curls, calf raises, leg press and leg extensions. The strength tests, blood samples, girths, and body weight will be performed before and after 4, 8, 12 and 16 weeks of resistance training. Height and muscle biopsies will only be performed before and after training has been completed.

**Risks:**

The exercise tests and training will require maximal physical and mental effort. However, the effort required will not be greater than that experienced during participation and competition in sport. As proper technique will be demanded throughout the study, they represent little risk to healthy, elderly individuals. After the strength tests you may feel some discomfort in your muscles which will disappear within a few days. The blood sampling will cause some discomfort and has a small risk of infection. Subjects are asked to report any abnormal effects (numbness, excessive pain and swelling) immediately.

**Time Commitment**

If you pass a medical examination you will then perform the following baseline assessments:

A one hour familiarization strength training session which will allow you to become familiar with the resistance training exercises that will be used in the study as well as gain an understanding in proper lifting technique.

Two (one hour each) upper and lower extremity maximal muscular strength assessments performed one week apart.

Five blood sampling sessions will take place during the 16 week study and each session should take approximately 20 minutes.

Training will require approximately 1.5 hours per day, 3 days a week for a total of 4.5 hours per week for 16 weeks or 72 hours for the whole project.

**Consent**

I acknowledge that I have read this form and I understand the test procedures to be performed and the inherent risks and benefits involved from participation in this project. I consent to participate understanding that I may withdraw at any time without prejudice. I may expect a copy of this consent form and report of my personal results after the study is complete. Any research publication as a result of this study will not identify you by name, and the data collected will remain in possession of the investigator to ensure confidentiality. I also understand that I may make any inquiries concerning any procedure that I do not completely understand. I consent to participate in this research project.

Name:_____ Signature:_____

(print) (sign)

Address:_____ Date:_____

_____

Phone:_____

Postal Code_____

Witness:_____ Investigator:_____

(sign) (sign)

# Appendix B

## Individual Data Plots

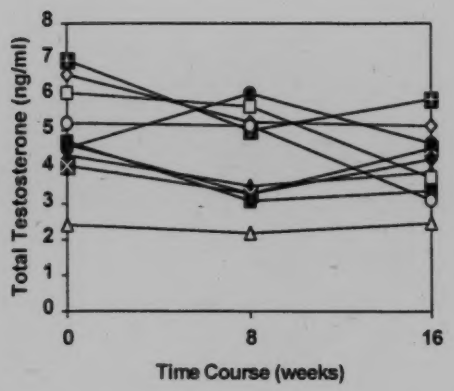



Figure B-1. Individual data plots of the TT concentrations of the RT group at 0, 8 and 16 weeks.

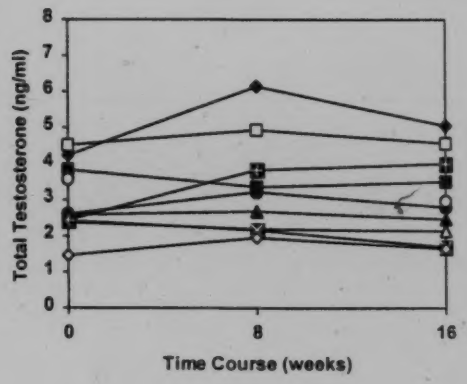



Figure B-2. Individual data plots of the TT concentrations of the CT group at 0, 8 and 16 weeks.

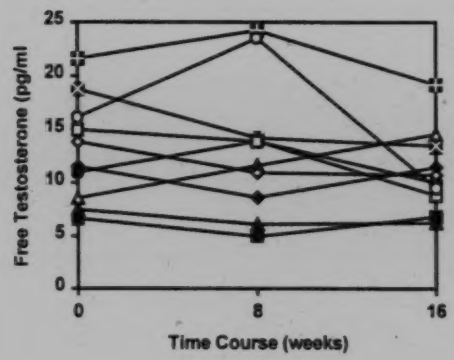



Figure B-3. Individual data plots of the FT concentrations of the RT group at 0, 8 and 16 weeks.

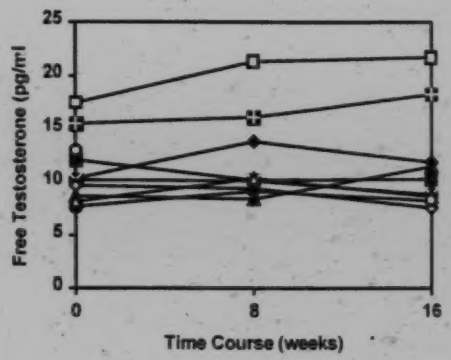



Figure B-4. Individual data plots of the FT concentrations of the CT group at 0, 8 and 16 weeks.

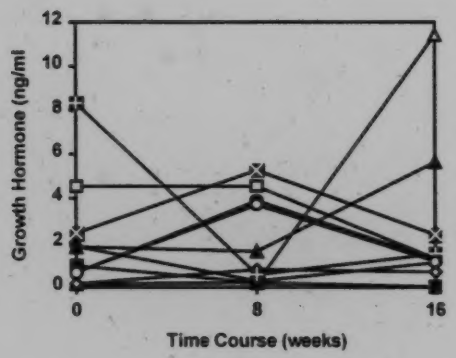



Figure B-5. Individual data plots of the GH concentrations of the RT group at 0, 8 and 16 weeks.

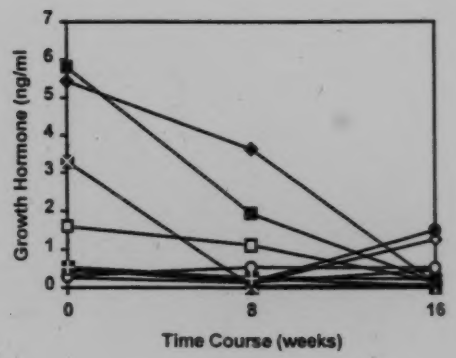



Figure B-6. Individual data plots of the GH concentrations of the CT group at 0, 8 and 16 weeks.

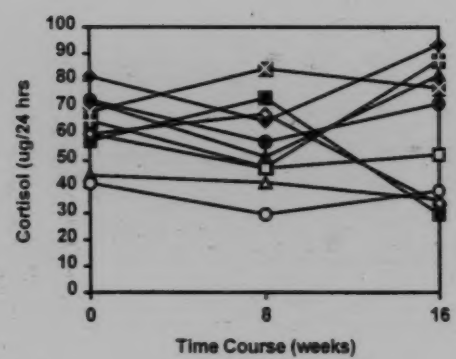



Figure B-7. Individual data plots of the 24-hour cortisol concentrations of the RT group at 0, 8 and 16 weeks.

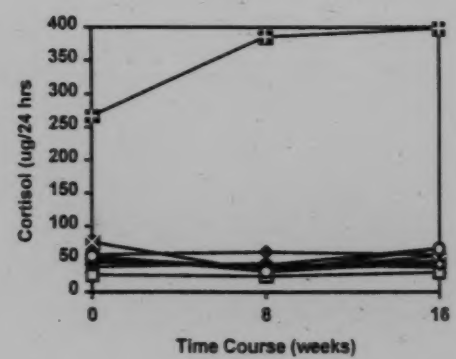



Figure B-8. Individual data plots of the 24-hour cortisol concentrations of the CT group at 0, 8 and 16 weeks.

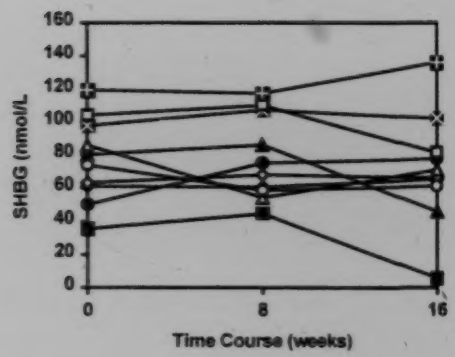



Figure B-9. Individual data plots of the SHBG concentrations of the RT group at 0, 8 and 16 weeks.

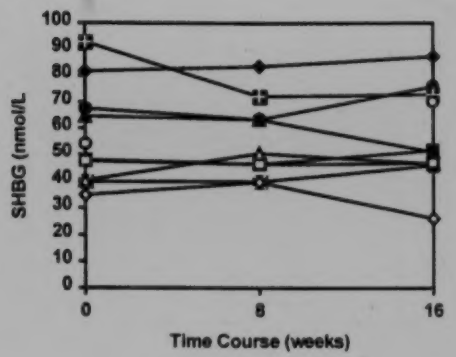



Figure B-10. Individual data plots of the SHBG concentrations of the CT group at 0, 8 and 16 weeks.

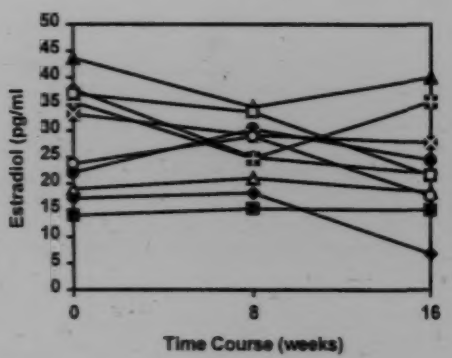



Figure B-11. Individual data plots of the estradiol concentrations of the RT group at 0, 8 and 16 weeks.

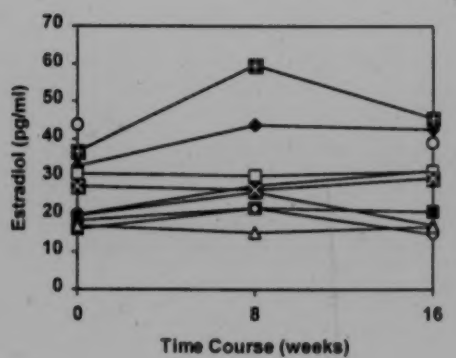



Figure B-12. Individual data plots of the estradiol concentrations of the CT group at 0, 8 and 16 weeks.